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I.

THE CUBICAL CAPACITY AND SUPERFICIAL AREA OF THE SPHENOID SINUS.*

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The sphenoid sinuses, although placed at the very center of the head, vary so greatly that it is incumbent upon those who are solving problems with reference to them, to study not only the relations which they bear to adjacent structures, but their form and size as well.

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In several papers already published, more particularly one on the "Relation of the Optic Nerve to the Nasal Sinuses,"[1] I discussed the linear measurements of the sphenoid sinus, that is to say, its anteroposterior, superoinferior, and lateral diameters, in fifteen heads. The variations in these thirty sinuses were as follows: Anteroposterior, 2 to 42 mm.; superoinferior, 4 to 38 mm.; lateral, 2 to 35 mm.

However interesting these facts are, elicited by careful reconstructions and accurate measurements, it must be remembered that the sphenoid sinuses are cavities, and hence present not only maximum diameters for study, but, far more, a surface covered with mucous membrane and a space enclosed by the walls of the sinus. It is to determine the area and the cubical contents of the sphenoid sinus and the possible relation between them that has impelled me to take up this study.

So far as I have been able to ascertain, no published report has been made of any observations looking towards the estimation of the area of the sphenoid sinus. In fact, little has been done in this particular, so far as any part of the nose is concerned. Dieulafé[2] measured the superficial area of the olfactory and the respiratory portions of the nose, respectively, by the laborious method of placing small squares of paper, 4 sq. mm. in area, one adjoining another, over the whole surface.

There has been more work done on the easier problem of determining the cubical capacity of the sinus. As far back as 1877, Braune and Clasen,[3] by the volumetric method, found the cubical contents of four sets of sphenoid sinus from corrosion preparations, as follows:

Head.	Right, cc.	Left, cc.
I	4.0	1.8
II	0.5	0.5
III	2.8	1.6
IV	6.5	6.9

Brühl[4] used an alloy composed of lead 8, tin 4, bismuth 15 and cadmium 3 parts, which melts at 65° C. After making casts of the sinuses in different heads, he then determined the

volume by noting the amount of displaced water. His results were as follows:

Head.	cc.
I	3.8
II	1.0
III	3.1
IV	1.6
V	1.9
VI	2.3
VII	1.8
VIII	1.8
IX	1.6
X	2.5
XI	2.1
XII	3.2

Important as these observations are, they lose much in consequence of the necessity for destroying the specimens in order to remove the casts. Hence, no study can be undertaken with the cast in situ.

The method which is here presented overcomes this objection, making it possible to obtain a mould of the sinus and at the same time to preserve the specimens for coincident or future study.

MATERIAL.

Heads that have been preserved by injecting a fifty per cent solution of formalin through the carotid were immersed in a three per cent solution of hydrochloric acid for three to five months. They were then sectioned horizontally by means of a sharp knife. Heads VII, VIII, IX, XI, XII, XIV have been described in a previous paper.[1] The remaining heads are here presented for the first time. They are from the dissecting room of the St. Louis University. All were adults, and in no instance has it been possible to ascertain any history of the individual.

METHOD OF MAKING PLASTER CASTS.

To obviate the destruction of the specimen in removing the mould, it was thought possible to use the method in vogue by dentists who take plaster impressions of the mouth and

teeth and patch any portion that is broken away in order to make them complete.

Plaster impressions were, therefore, taken of that portion of the sinus lying within the various adjacent sections, and these were then united at the proper place, resulting in a complete mould of each sinus.

PLASTER CASTS.

The plaster casts, twenty in number, give a clear idea of the size, form, variety and irregular contour of the sinuses.

The illustrations accompanying this paper show (a) the casts placed in the lowermost section of the corresponding head, with a view of graphically showing the extent of the cavities, (b) an anterior view of the right and left sinus of each head, (c) a superior, (d) external, and (e) internal view.

The exact relation of the two sinuses to one another is shown in each instance except in the external view, in which it would be manifestly impossible.

GROSS APPEARANCE.

When the plaster casts of the sinuses are placed in the most inferior of the horizontal sections in which the sinuses are found, their extent with reference to adjacent structures can easily be seen. The outlines of the latter are drawn faintly, so as to bring out the sinuses more forcibly.

Head VII. The two sphenoid sinuses project far back and lie closer to the torcular than those of any other head. All the sinuses of this head were unusually large. External wall most irregular. Anterior wall reaches a lower level of the right than that of the left.

Head VIII. The sphenoids are fairly small and regular. The section shown is quite thick, and more than half of the sinuses are concealed. All walls fairly smooth. External walls triangular in shape.

Head IX. Sphenoids, both small, project superiorly beyond the adjacent ethmoids. The larger portion of the sinuses lies above the section. All walls irregular in contour and more or less quadrilateral in shape.

Head XI. Very similar to head IX, except that sinuses are larger, especially in superoinferior direction. The shape is that of a prism.

Head XII. The left sphenoid overshadows the right, which appears as only a slight projection from its inner and anterior wall. It is in relation with the optic chiasm and both optic nerves, as well as the posterior ethmoid cells on the right side. The operative difficulties which such sphenoids might occasion can be well appreciated.

Head XIV. Right sphenoid much larger than the left, in relation with optic chiasm and both optic nerves. While the left sphenoid is of fair size, the right overtops it and extends some distance to the opposite side. External wall of the right sinus rhomboidal in shape; left sinus is a prism with rounded ends.

Head XXIII. Both sphenoid sinuses fairly uniform, having about the same relative extent. Larger portion lies above the section shown. Both quadrilateral in shape.

Head XXVI. Right sphenoid sinus much the larger, in relation with optic chiasm. A mere section gives no idea of relative size. Left, rhomboidal in shape; right, rhomboidal laterally, but otherwise more rectangular.

Head XXVII. Both sphenoids large and fairly uniform. Section shown contains the larger portion. External wall very irregular, but fairly rectangular in form.

Head XXXV. Both sphenoid sinuses very large, especially in the lateral direction. However, they are quite similar in size and extent. All the walls are triangular and very nearly equilateral.

It will thus be seen that few of the sinuses in the different heads resemble one another in size or form. While the two sinuses in an individual head may be similar, they are in the main different from those of another head. (Heads VIII, IX, XXIII, XXVII.) The greatest difference in size and form in a given pair is to be found in head XII. The difference is somewhat less in heads XIV and XXVI. Both sinuses in heads VII and XXXV are immense, while those in head XXVII, and the right sinus in heads XXVI and XIV, and the left in head XII are quite large. A great difference in size is to be noted between the right sphenoid of head XII and the left of head XXXV. The fairly smooth and regular contour of the sinuses in head VIII may be well contrasted with the projections and concavities and irregular shapes of the sinuses in heads VII and XXXV.

CUBICAL CAPACITY.

The measurement of the displacement of water resulting from the immersion of a body in water gives the volume or cubical capacity.

As the casts are porous, they were first dipped into melted paraffin, so that the interspaces might be made impervious to water. After this the cubical capacity was ascertained volumetrically in cubic centimeters, as follows:

Head.	Right. cc.	Left. cc.
VII	6.5	9.9
VIII	3.8	3.0
IX	2.4	2.3
XI	4.0	4.5
XII	0.6	7.5
XIV	6.3	1.0
XXIII	3.0	3.1
XXVI	11.8	1.9
XXVII	4.8	8.2
XXXV	8.3	10.0
Average	5.15	5.14

Average of all the sinuses.....5.145

The range is from 0.6 to 11.8.

These figures are greatly in excess of those of Braune and Clasen, with an average of 3.08 cc., and of Brühl, with an average of 2.23 cc. This may be accounted for by the fact that in the former the range was 0.5 cc. (of which there were two) to 6.9 cc., and in the latter from 1.0 cc. to 3 cc. Taking all together, the average for the forty sinuses is 3.73 cc.

The similarity in the averages of the right and the left sphenoid sinuses in the ten heads is most striking. It would be unfair, however, to draw any definite conclusion from this until many more such sinuses are subjected to the same method of measurement.

SUPERFICIAL AREA.

Various experiments were made to discover a suitable method for determining the superficial area of the cavities. It was soon established that a fair estimate could be made by

taking a given quantity of material, cutting it appropriately and fitting the various pieces over the plaster cast so as to cover it completely without any overlapping.

The superficial area equals the known quantity less the amount remaining, which can be easily measured by the use of paper ruled in square millimeters.

Tin foil was first used, but it was discarded because it failed to adhere sufficiently to the casts. Gummed paper was then utilized, but it failed to adhere satisfactorily. However, it was found possible to color the area covered by each slip of paper with pencil marks until no portion of the cast remained free.

Finally, adhesive plaster was found most acceptable. A strip equalling 25 square centimeters was placed with the adhesive part down on a small board without stretching. Pieces were cut with a knife and fitted upon the cast until it was entirely covered, without any overlapping. Though this was somewhat laborious and tedious at first, it soon became less difficult and consumed less time.

It is safe to say that this method is not subject to an error of two per cent; in fact, it is more than likely that there is not more than a half of one per cent of error.

The results in square centimeters are as follows:

Head.	Right. qcm.	Left. qcm.
VII	26.7	27.2
VIII	13.9	12.9
IX	8.9	9.7
XI	13.2	16.5
XII	2.4	22.7
XIV	24.7	4.0
XXIII	10.5	12.2
XXVI	26.4	7.4
XXVII	17.3	21.8
XXXV	25.5	28.2
Average	16.95	16.26
General average		16.65

Here again is found the remarkable similarity in the average of the right and of left sphenoid cavities. A larger number of

observations will be necessary to establish whether or not this is the rule.

Having established the cubical capacity or volume and the superficial area of 20 sinuses, it was thought desirable to determine a formula by which it would be possible to estimate the superficial area from a known cubical capacity. It is not difficult to determine the cubical capacity of a number of sphenoid sinuses, but the superficial area is a much more difficult undertaking. For this reason such a formula is of value.

The data given result in the formula

$$Y = 0.2\ X + 4.4.$$

X is the volume and Y is the superficial area divided by the volume. In order to determine the superficial area, the value of Y must be multiplied by the already known volume of the sinus. The curve, figure 31, corresponds very closely to the figures for the sinuses measured.

Dr. Virgil Loeb made the casts for me, Mr. Tom Jones the drawings, and Leo Loeb, M. E., the formula.

CONCLUSION.

The casts prepared according to the method described shows that the cubical capacity in the twenty sphenoid sinuses examined varies from 0.6 to 11.8 ccm., with an average of 5.145 ccm.; and the superficial area from 2.4 to 28.2 qcm., with an average of 16.65 qcm.

The formula for calculating the superficial area from the known cubical capacity is subject to change, depending upon additional data.

LITERATURE.

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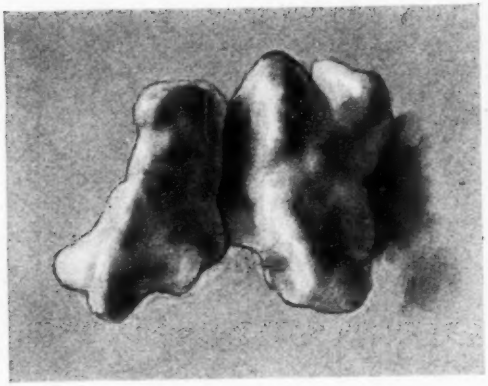
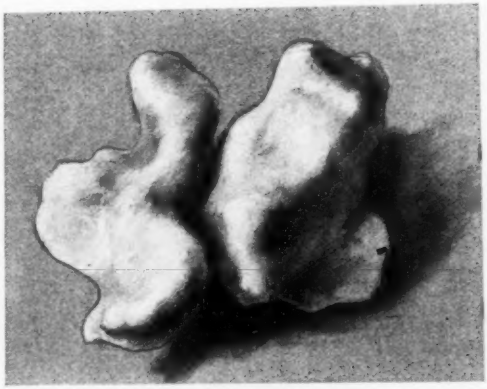


HEAD VII. Anterior view.



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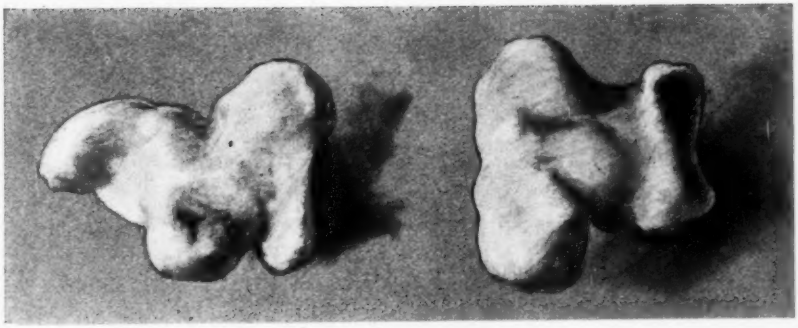

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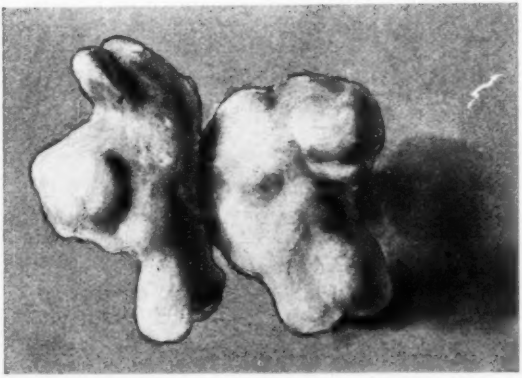

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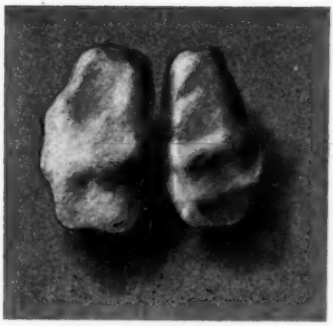

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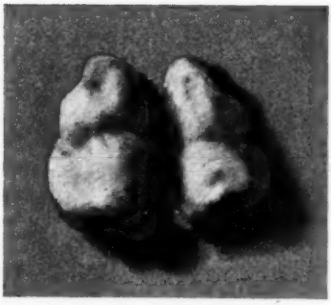
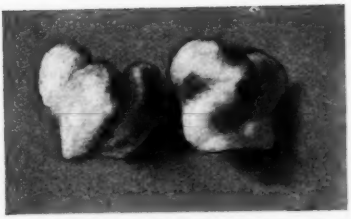


HEAD VIII. Superior view.

HEAD VIII. External view.

HEAD VIII. Inferior view.

HEAD IX. Anterior view.



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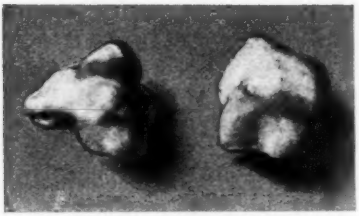

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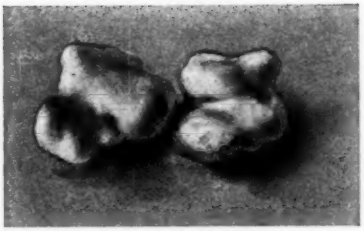

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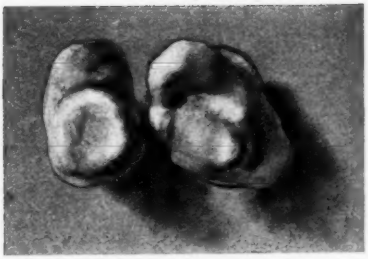

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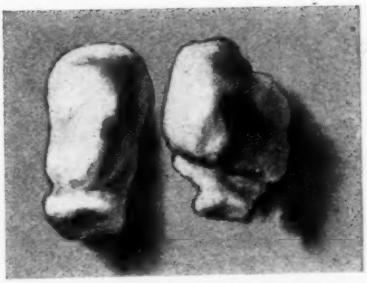

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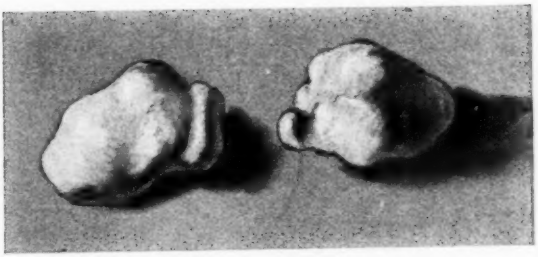

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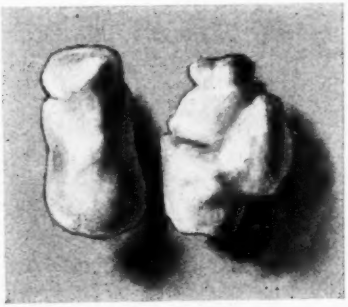
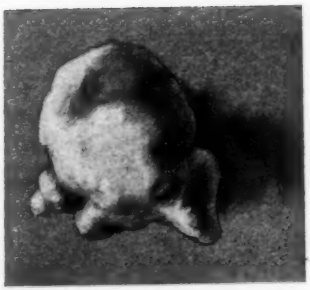


HEAD XI. Inferior view.

HEAD XII. Anterior view.



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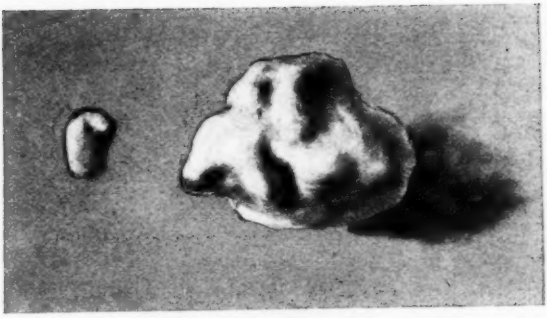

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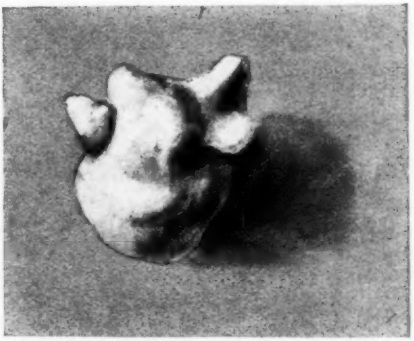

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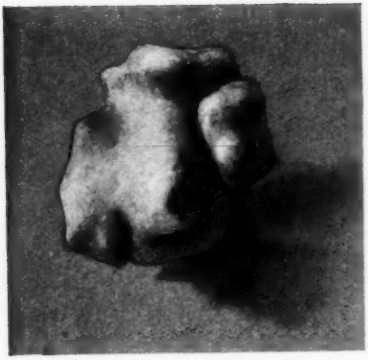

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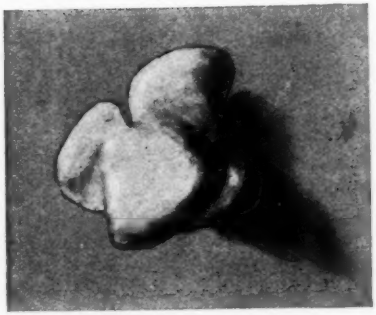

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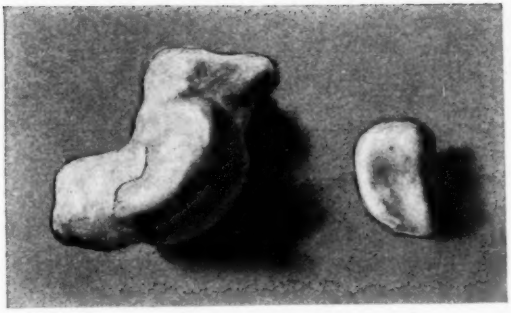

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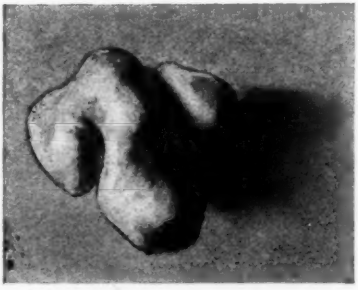

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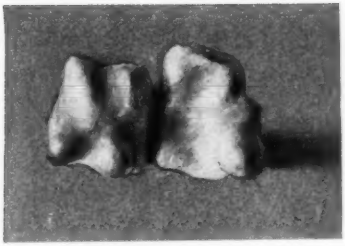

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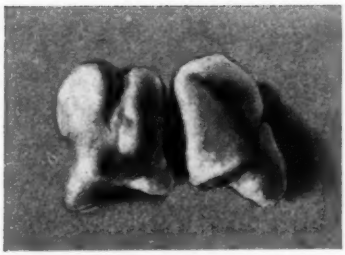

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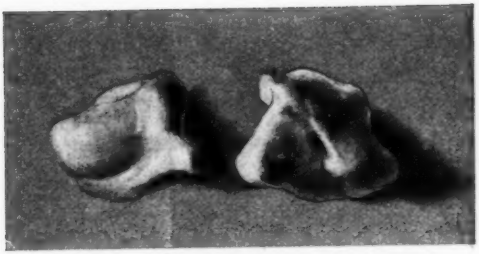

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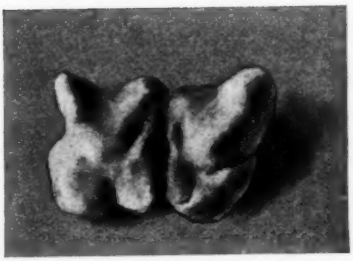

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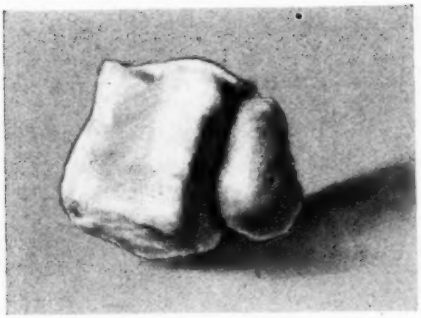

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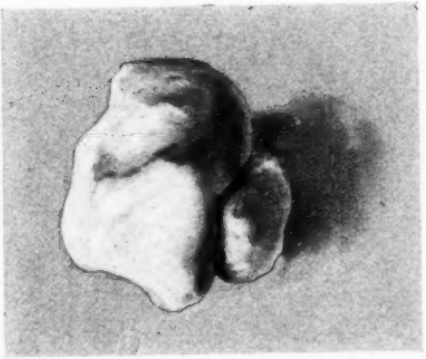

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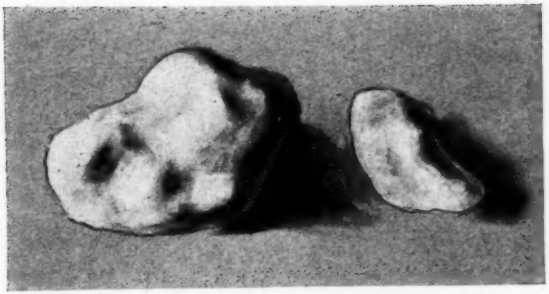

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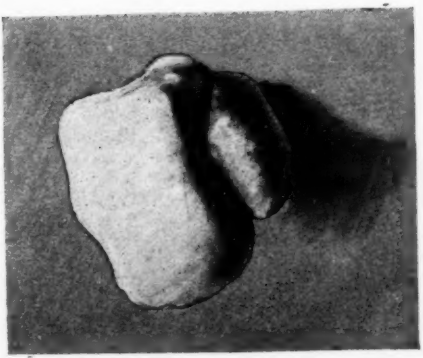

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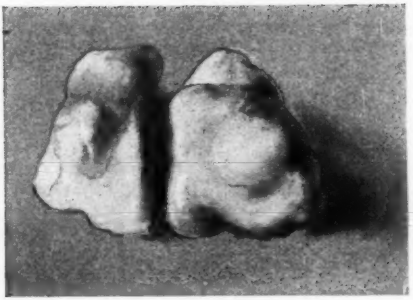

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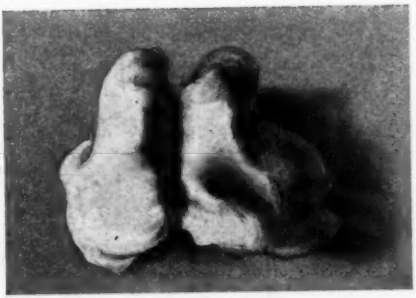

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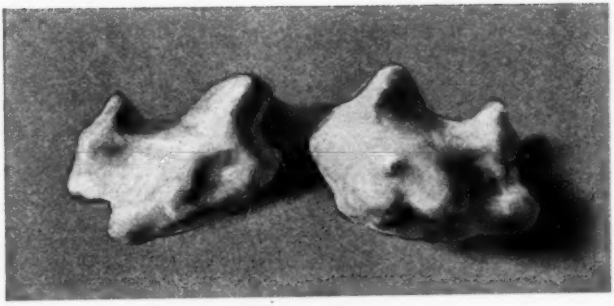

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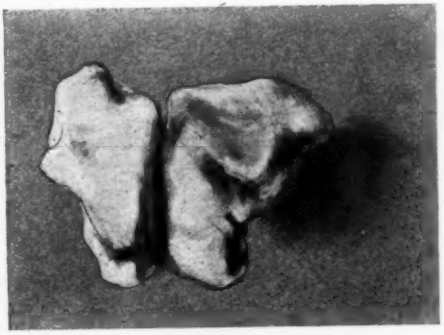

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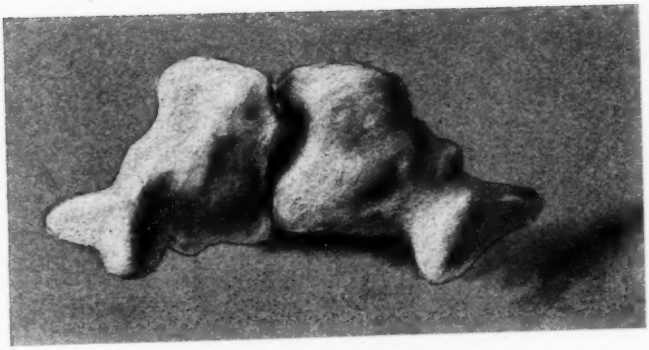
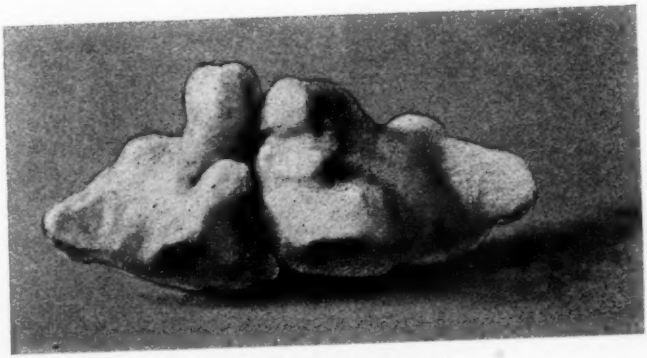
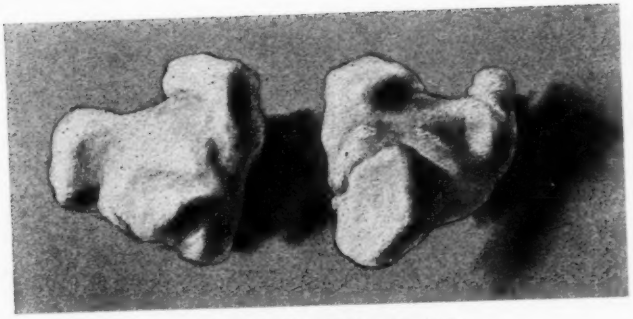


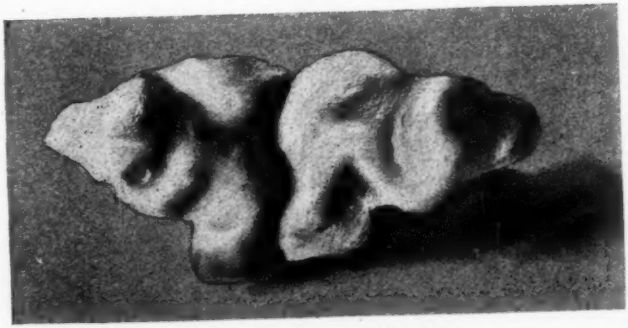
HEAD XXXV. Anterior view.



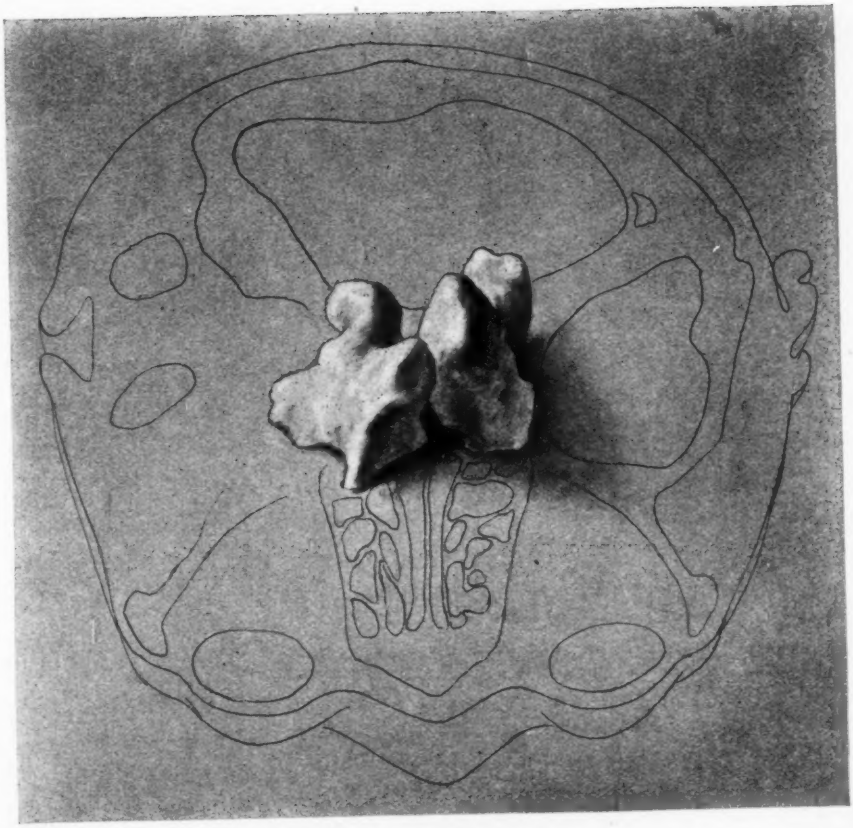
HEAD XXXV. Superior view.



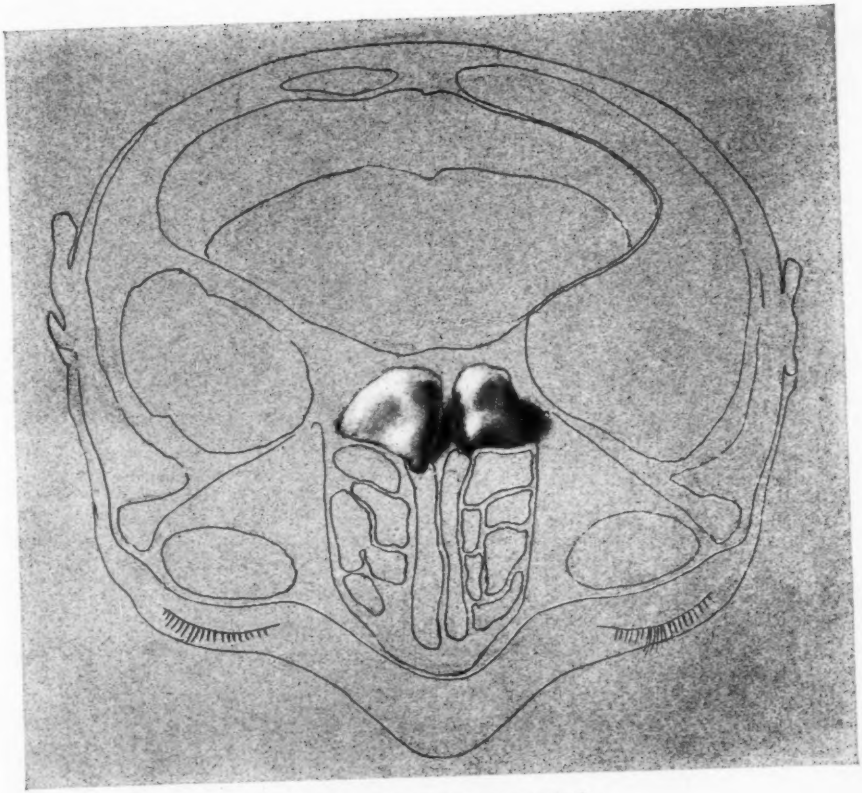
HEAD XXXV. External view.



HEAD XXXV. Inferior view.



HEAD VII. Reduced one-fourth.



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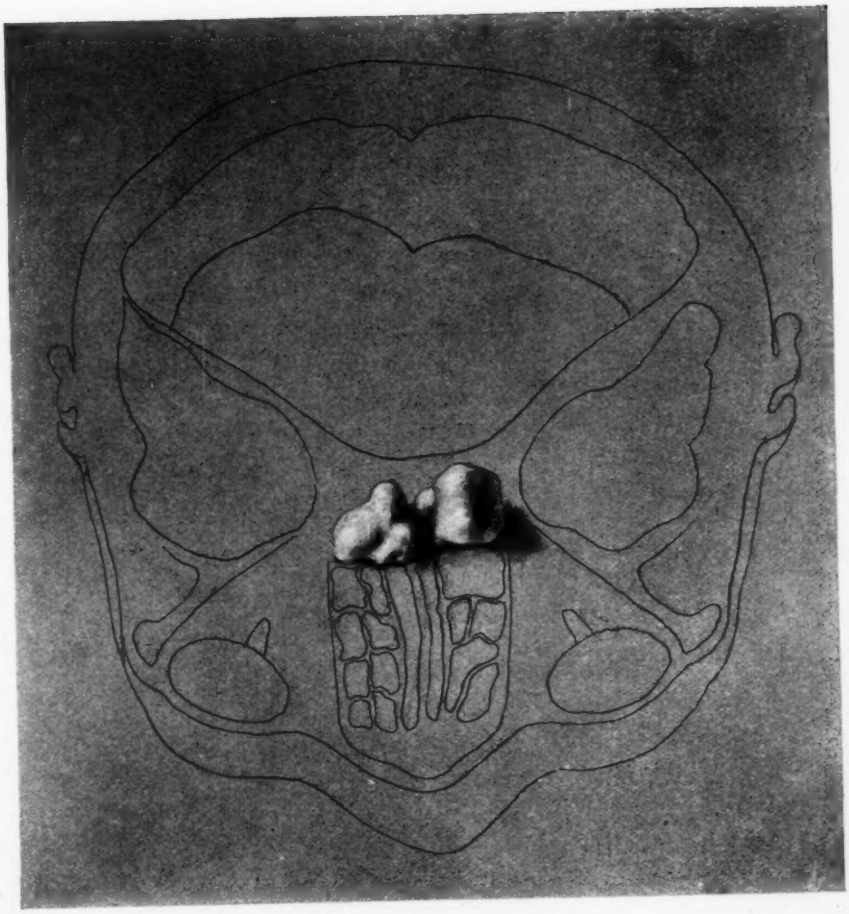

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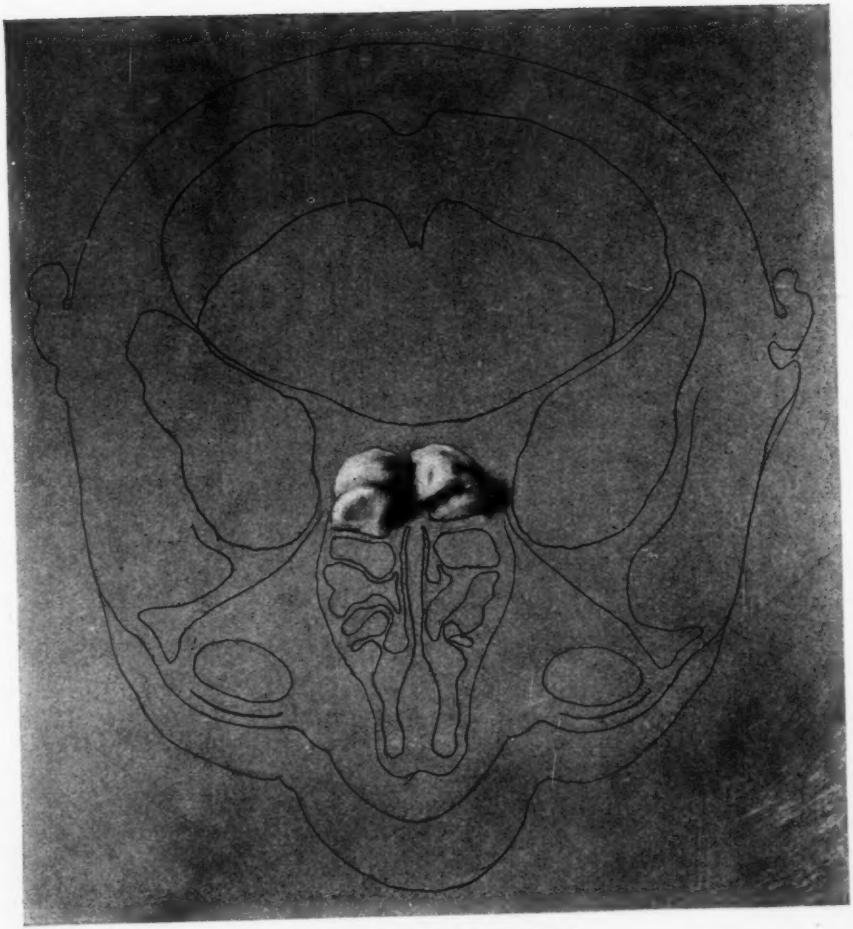

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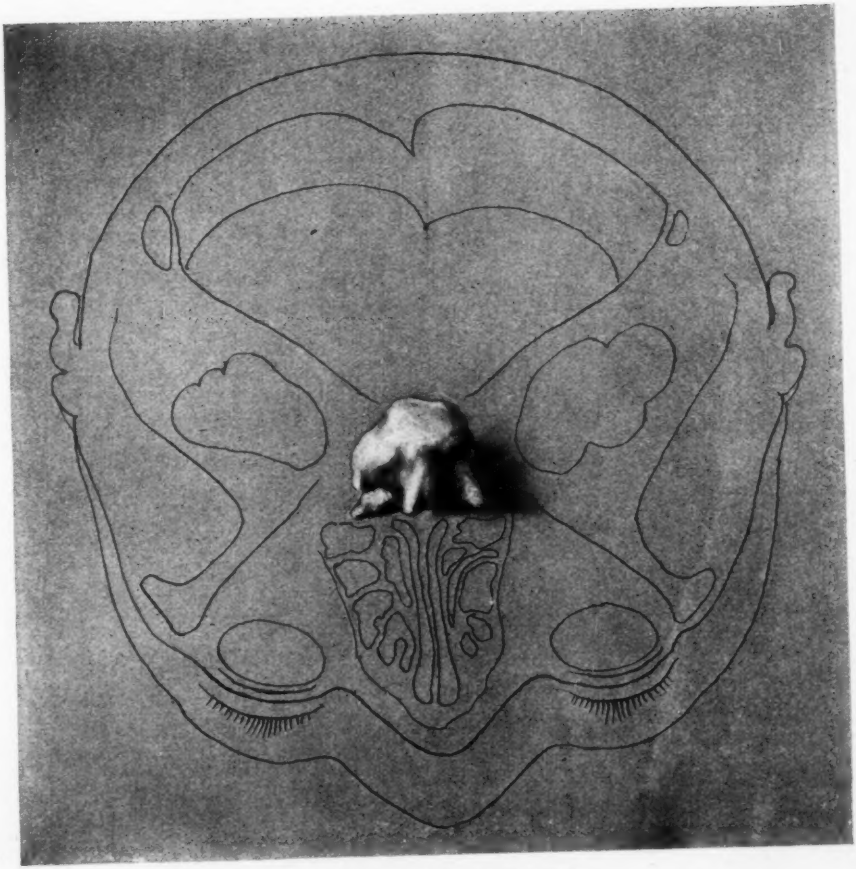

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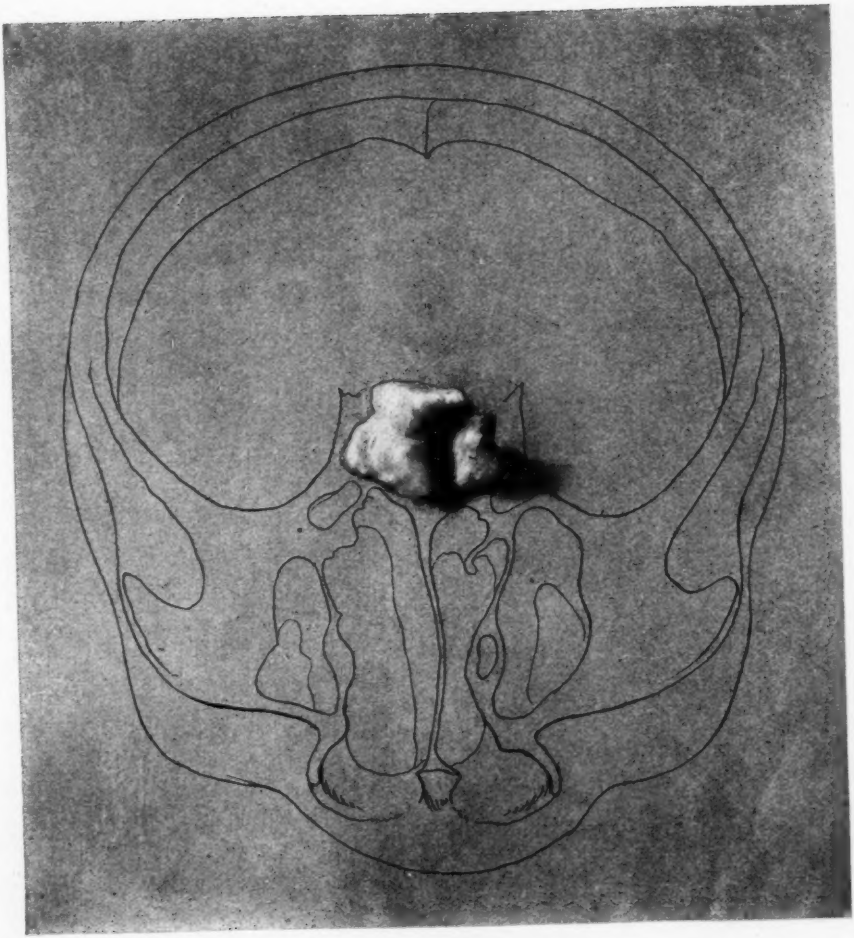

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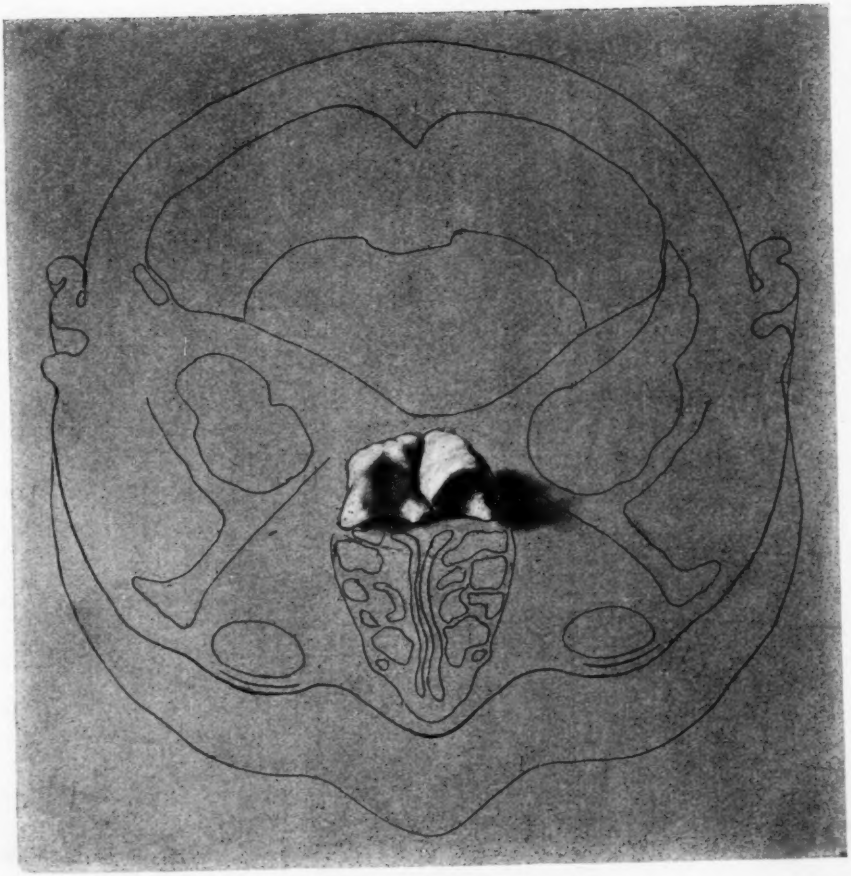

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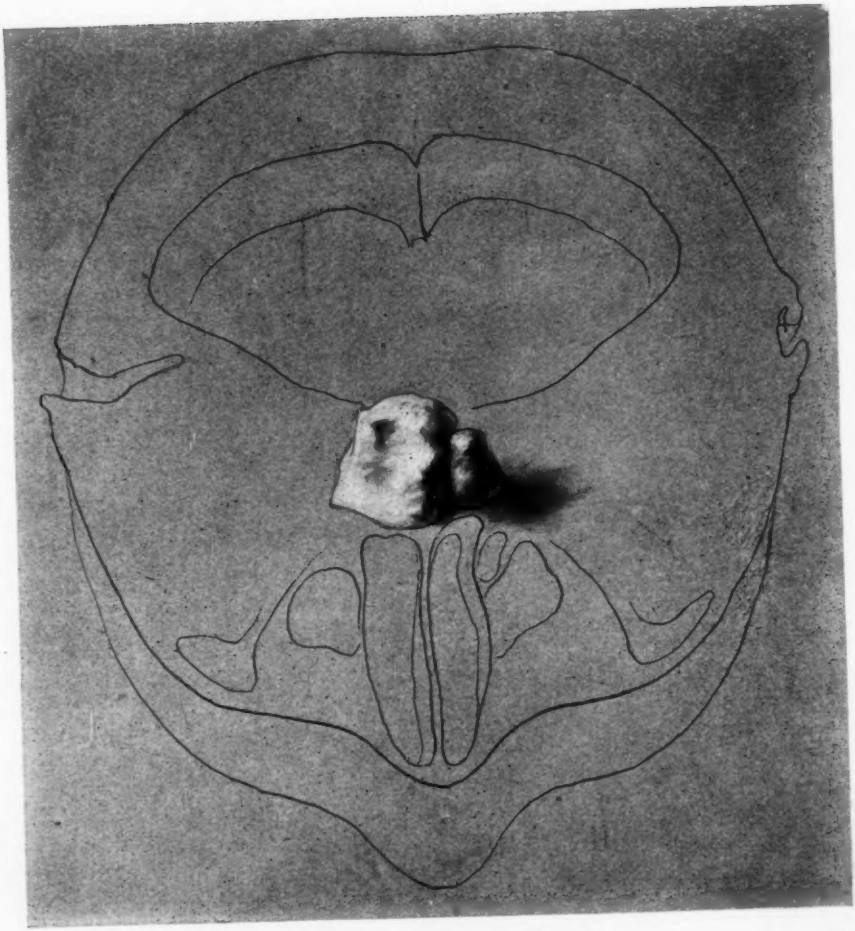

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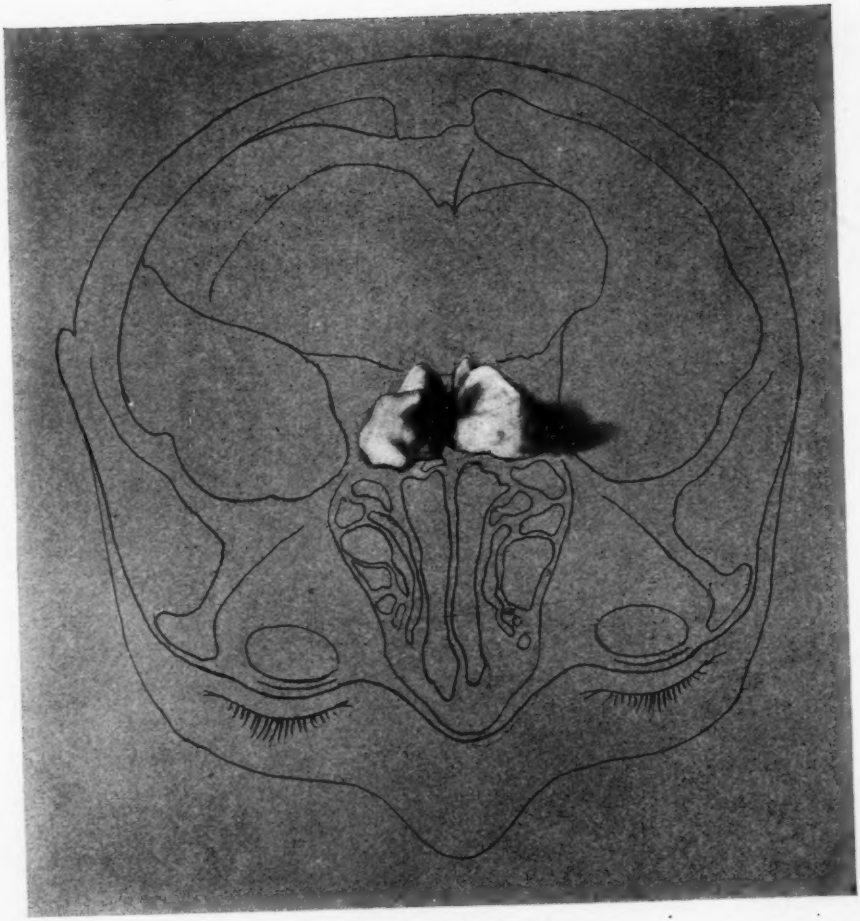

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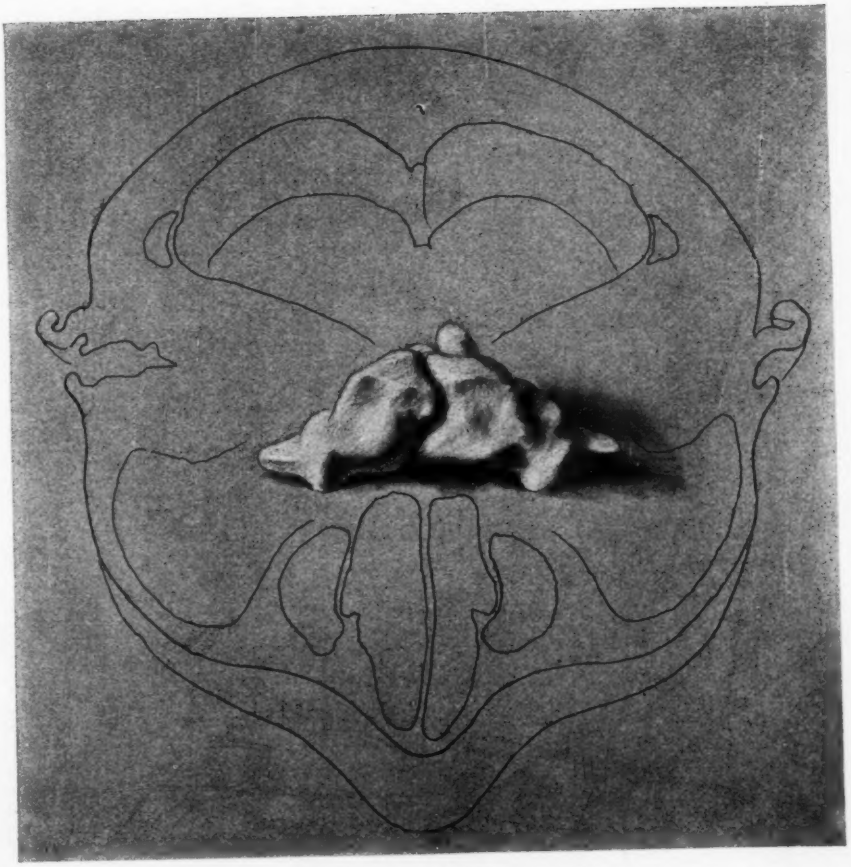

HEAD XXXV. Reduced one-fourth.

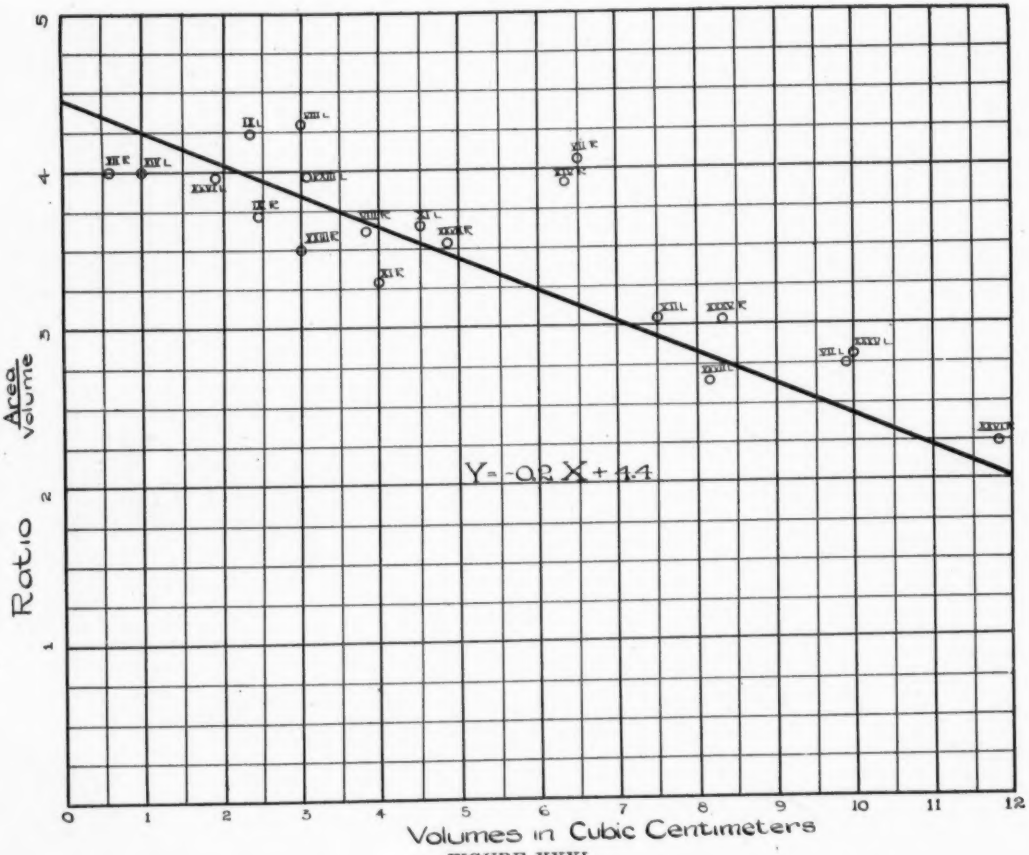

FIGURE XXXI.

Curve of formula for determining the superficial area, the cubical capacity being known.

II.

FUNCTIONAL TESTING OF THE VESTIBULAR APPARATUS.*

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Vienna.

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New York.

ANATOMY AND PHYSIOLOGY.

In the discussion of the anatomic and physiologic relations of the semicircular canals, utricle and saccule, I do not intend to make a complete anatomic and physiologic or even historic presentation, because this may be found in the text books and monographs already published by Ewald, von Stein, Wanner and Bárány. I wish rather to give special consideration to the questions still to be solved.

(*a*) *Position of the Semicircular Canals.*

Concerning the position of the semicircular canals in the skull and with respect to each other, it has always been taken for granted that the relations here are very constant, but Schönemann, by means of corrosive preparations of the labyrinth from a great collection of skulls, has shown that the individual variations are considerable. The angle which the plane of the horizontal semicircular canal makes with the horizontal plane varies from zero to thirty degrees. In other words, in many cases the horizontal canal lies, when the head is in an upright position, in a horizontal plane; in other cases, inclined even to thirty degrees backward, downward

*Read before the German Otological Society at the Congress held in Frankfort on Main, June 2nd and 3rd, 1911.

and outward. The angle which the anterior vertical canal includes with the median plane varies more than thirty degrees, viz., between thirty and sixty-five degrees. Between the right and left sides differences of twenty degrees are found. The angle which the canals make with each other is by no means always a right angle. Thus the angle between the plane of the anterior vertical and horizontal canals varies between sixty-five and ninety degrees; the angle between the plane of the anterior and posterior vertical, between eighty-five and one hundred and fifteen degrees. Most constant is the angle between the horizontal and posterior canal—ninety to one hundred degrees, but in most cases exactly ninety degrees.

Unfortunately, no efforts have been made on a living human being to determine the planes of the semicircular canals with certainty. For the functional tests several important points are to be derived from the observations of Schönemann. While working out the caloric test it struck me from the beginning that, with the upright position of the head in different individuals, the nystagmus is sometimes more horizontal and less rotary, sometimes almost entirely rotary. If we take into consideration the position of the horizontal semicircular canal, we may expect that where the horizontal canal is exactly in the horizontal plane, with the head in an upright position no horizontal nystagmus will arise upon caloric irritation. Where, on the contrary, this canal is inclined backward, outward and downward, horizontal nystagmus must become manifest upon application of the caloric test. The following problems, therefore, appear of significance to me: Are the central connections of the canal nerves always the same in different persons upon different positions of the canals themselves? Does the motion of the endolymph in a canal really always produce eye movements exactly in the plane of this canal? Let us again consider the horizontal canal. If it is lying in the horizontal plane with the head in an upright position, then upon turning the patient about the vertical axis, pure horizontal nystagmus should be called forth; but if the horizontal canal is inclined thirty degrees to one side and backward, then with the same central connections and with upright position of the head, turning about a vertical axis could not call forth a pure horizontal nystagmus, and such a nystagmus could arise only by forward inclination of the head thirty degrees.

(*b*) *Structure and Physiologic Excitation of the Nerve Ends in the Canal.*

The sense organ of the ampulla of the canal is situated upon a hill of cells transversely to the longitudinal axis of the canal, called the crista ampullaris. The sense cells cover this entire mass from the base to the summit, interrupted by the supporting cells. In many animals there is found on the summit of the hill a zone in which no sense cells are present. In man such an indifferent zone is not found. Every sense cell possesses several hairs which are two or three times as long as the cell itself. These hairs enter, after a short free course (about a half cell length), into the cupola and run in fine canals in the same for a short distance. The hairs at the base of this cell eminence incline toward the lumen of the canal. (Breuer.) Upon cross section of the cupola the canals in which the hairs of the sense cells lie are distinctly seen to reach still further upward toward the upper margin of the cupola, which itself is two or three times as high as the hairs. The entire construction of the cupola gives the impression that there is here a continuously renewed mass which evidently becomes separated from the supporting cells and crumbles off at the upper free margin. (Breuer, Kolmer.) Thus it is explained why the canals in the cupola reach out further toward the free margin than the cell hairs.

The distribution of the nerves in and around the sense cells is also interesting. Cajal asserts that the distribution of the nerves concerned in the formation of the crista ampullaris is not everywhere the same. The strongest and richest fibers are distributed to the sense cells which are found near the summit of the crest. Fewer nerve fibers go to the slopes of the hill. This speaks perhaps for the greater functional value of the cells at the summit of the crest. Kolmer, Brühl and Bielschowsky have demonstrated recently that the nerve fibrils end ultimately in the sense cells, while Cajal does not admit this. This question is of great histologic importance, but has little interest for otologists; for it is to us of little moment whether the irritation which meets the sense cell is transported by contact or through direct transmission along the nerve fibrils. The following facts are of greater significance to us: Cajal has demonstrated that the nerve fibers

form plexuses around the sense cells and that one fiber is in connection with several sense cells. Kolmer, Bielschowsky and Brühl have also seen anastomoses between the nerve fibers. Kolmer observed further that one and the same cell can be supplied by two different nerve fibers. These facts are to be understood from the function of our sense organ. While in the retina two sense cells lying side by side can receive different impressions, in our organ a greater number of sense cells always receive the same excitation. For the uniform distribution of this impulse to many sense cells and nerve fibers it is certainly of advantage if the described plexus—and anastomoses—formations are present.

We will now try to imagine how an impulse occurs in the ampulla of the canal. As we all probably now assume with Breuer and Mach, the excitation of the ampulla is due to an endolymph movement which strikes the cupola and displaces it a little out of its resting position. The amplitude of this movement can only amount to a hundredth part of a millimeter or less. Greater displacements would separate the cupola from the hair cells, which indeed happens as a result of strong endolymph disturbances. This results, for example, upon division of a canal. (Breuer.)

Breuer has a rather distinct idea of the kind of influence which the displacement of the cupola exerts upon the nerve cells, and I should like, in spite of the extremely hypothetical nature of all these considerations, to discuss this thoroughly, since our views may perhaps be cleared up thereby and new investigation may thus be also stimulated.

Breuer assumes that an endolymph movement which displaces the cupola toward the utriculus stretches the hairs upon the canal side of the crest slope and relaxes the hairs upon the utricular side. But in man there are also other hair cells present at the summit of the crest. These, it appears to me, must be stretched just as much by displacement toward the utriculus as away from it. We shall come back to that later. Breuer assumes that only the stretching of the cell hair works as an irritant. It seems possible that the relaxation has also a function, viz., the inhibition of rest tonus of the sense cells analogous to that produced in the otoliths of organs through gravity. To this end two conditions would be necessary: first, that the cupola has not the same specific weight as the sur-

rounding endolymph; second, that the difference of the specific weight is sufficiently great in order that the influence of gravitation may produce an irritation which crosses the threshold of excitability of the sense organ. There are investigations which go to prove that the difference of specific gravity of the cupola in comparison with that of the endolymph is very trifling, viz., the rotation tests of Wittmaack. Wittmaack turned guinea pigs about two thousand times in a minute—i. e., about thirty-four times a second—in a centrifuge. As a result, the otolith membranes were torn off; but the cupolæ remained in correct position. One might consider this a demonstration that the cupolæ themselves have the same specific weight as the endolymph, and I myself in the beginning fell into this error. One must, however, remember that the firmness of the connection also plays a role. Only when the given centrifugal force becomes greater than the firmness of the connection can the cupola be torn off. Further investigations by more rapid turning must be carried out to decide this question.

Whether or not there exists a tonus of this kind going out from the ampullæ is certainly not of great physiologic significance; for, according to their entire structure, the otolith organs are surely better adapted to this end. Moreover, the tonus must be also of central origin. For the sake of theory the solution of this question would be full of significance, since the excitation through gravity and through progressive movement in the semicircular canals and nerve end places of the saccule and utricle would give no principal but only a quantitative difference. Both end places would functionate in the same way, only the otoliths would participate more strongly. The excitation through progressive movement performed in the ampullæ of the canals in the beginning of the movement would allow the cupola to remain behind as a result of its greater specific weight, as compared with the endolymph.

The chief function of the ampullæ, however, remains, viz., to become excited during turning. Ewald has demonstrated that according to the direction of the endolymph movement the effect is of varying degree even when the mechanical irritation is the same. Thus in the horizontal canal the endolymph movement toward the ampulla brings about a significantly stronger (almost double as strong) reaction as the movement toward the smooth end. This is reversed in the

vertical canals. An explanation of this fact has hitherto not been given. I believe it may lie in the distribution of the nerves in the crista ampullaris. If we assume that more than half of the nerve cells of the crista of the horizontal ampulla are destined to become excited through the jolt toward the utriculus, then everything could be understood. Thus would also be explained the function of the nerve cells situated at the summit of the crest. As already mentioned, it looks as if these cell hairs would become stretched by every endolymph jolt toward the ampulla as well as away from it.

Let us now assume that the cell hairs at the summit are all destined for the reaction due to endolymph motion toward the utriculus. Then it is clear that the reaction produced by movement toward the ampulla must be the stronger. If, however, the endolymph jolt is exerted away from the ampulla, then the following happens: The hairs on the utricular side now become stretched and produce in their central connections correspondingly contrary reactions. But the hairs of the cells at the summit of the crest are also stretched. This excitation works diametrically opposite to the irritation of the hair cells on the utricular side and brings about a further weakening of this reaction. It is even possible that with a certain distribution, a complete inhibition of the excitation takes place in the center. At any rate, there would be explained by varying distribution of the nerves in different individuals, variations in the effect of the endolymph movement toward the ampulla and away from it.

The question whether a rest tonus exists in the end organs of the ampullæ is followed immediately by the question as to how long the irritation in the ampulla upon turning lasts. At this point we will anticipate the following statements, viz., that the beginning of the turning movement brings about a turning sensation in the direction of the turning and a nystagmus in this direction. Let us consider the horizontal canal and an endolymph jolt which moves the cupola toward the utricle, as, for example, in the beginning of a turning toward the right. We distinguish a canal side and a utricular side of the ampullary crest. Breuer imagines the following: through the displacement of the cupola the hairs on the canal side of the crest are pulled and bring about the sensation of turning toward the right. (Concerning nystagmus of the eyes and of the head,

Breuer does not speak.) If turning toward the right be continued at the same rate of speed for a longer time, then the cupola becomes gradually drawn backward again into its resting position, as a result of the elasticity of the hairs and probably of mucous drops. As long as the resting position is not reached, the turning sensation toward the right continues. If, now, the resting position is attained with the same jolt with which the movement began, then the displacement of the cupola toward the canal side results from the endolymph wave directed from the ampulla to the smooth end. This reversed displacement brings about naturally the contrary turning sensation, which again lasts until, upon quiet position of the head, the cupola has been brought back into its normal position through its elastic strength. If, however, the turning does not last so long as the return of the cupola into its normal position demands, then the following happens: First, there results again the push which displaces the cupola toward the utricle. However, before a retraction of the cupola worthy of the name has taken place, through the elasticity of the hairs, there ensues the opposite movement through stopping, in consequence of which the cupola now becomes again immediately displaced back into its normal resting position, and there arises, therefore, after a turning of such duration, no after-sensation upon stopping. Against the theoretical side of Breuer's statement there is nothing objectionable, in my opinion, especially not if one assumes as fact a constant centripetal tonus from the sense cells of the ampulla. If there does not exist a tonus of this kind, then it would seem credible that the outgoing excitation from the sense cells lasts a shorter time than the displacement of the cupola, while during the still existing trifling displacement the irritation vanishes. (Abels.)

But how are the facts related to Breuer's hypothesis? If we turn a great number of normal persons, then we may observe the following facts: The turning sensation after turning lasts in different persons, even in so far as possible with the same kind of turning, varying lengths of time. While one has a turning sensation of only a few seconds, another has this sensation for forty-five seconds or longer. In both cases a nystagmus of the eyes may be of the same strength and of the same duration. Breuer has taken no account

of the nystagmus of the eyes and of the head in his work. That is, however, absolutely necessary; for the nystagmus is indeed a much more certain sign of excitation of the canal apparatus than the turning sensation, and has the advantage that it is accessible to objective investigation. The fact that in two cases the nystagmus may last the same length of time but the turning sensation unequal lengths of time, overthrows Breuer's hypothesis. It is incredible that the peripheric condition of the sense organ alone has to be called to account for the duration of the turning sensation. Even if we had to assume with Breuer that so long as the cupola is pushed aside an irritation is produced in the sense cells, then the question of the duration of this peripheral irritation is not yet decided.

It is a matter of consequence how the center reacts to this irritation. There are people whose turning sensation vanishes even while the nystagmus continues, and others who have this sensation about as long as the nystagmus persists. The question follows whether the duration of the nystagmus can be explained by occurrences in the peripheral sense organs. However, this is also not possible. I should like to introduce one certain fact. Let us turn a man ten times and then stop suddenly; there follows, let us suppose, a nystagmus of one and a half minutes' duration. If we turn the same man thirty times and then stop again suddenly, the after-nystagmus lasts only forty seconds. This fact alone demonstrates that even in the duration of the nystagmus central relations play an important role. If we take only the nystagmus into consideration, as a result of the following observations, then we can say that the strength and duration of the nystagmus is certainly dependent in part upon the strength and duration of the peripheral irritation, but the excitation of the centers in which the nystagmus is produced plays a further very important role. If, for example, the excitability is diminished, then the same peripheral irritation brings about a weak nystagmus of only short duration, and upon increase of the excitability this same irritation has, as a result, an extraordinarily strong and long-continued nystagmus. If it comes, as in head movements of short duration, to an immediate cessation of the peripheral irritation, then the centrally produced nystagmus may thereupon immediately be stopped, and there arises upon

stopping no after-nystagmus. Under pathologic conditions, however, even with an intact peripheral sense organ, the peripheral cessation of the irritation cannot suffice to inhibit the suddenly released central forces, and there arise then attacks of dizziness which are produced through quick head movements. Also in caloric nystagmus there are conditions which prove the function of the centers. If we irrigate the right ear of a normal person with cold water, head in the upright position, there arises horizontal and rotatory nystagmus toward the left. If we incline the head 90 degrees toward the left shoulder, then this nystagmus is transformed into a horizontal nystagmus toward the right. In many cases, however, this transformation is wanting. (Bárány, Hofer.) This can only be explained by the fact that the change of the endolymph movement is not strong enough an irritant to inhibit central nystagmus directed to the other side and to produce the corresponding nystagmus toward the right. It is much easier for the transformation of the rotary and horizontal nystagmus toward the left to occur by inclination of the head toward the left, because for this transformation a much smaller amount of central work has to be done.

(*c*) *Structure and Physiologic Excitation of the Nerve End Places of the Otoliths.*

In his fundamental work concerning the function of the otoliths, Breuer gives an anatomic description of the sense organ which interests us here. As for the mammalia, he states that the macula of the utricle and that of the saccule are in vertical relation to each other, as Rüdinger first described it. Breuer says: "The important and unequal shrinking of the cartilage forces us to greater reserve, if we try to determine whether and in what direction the maculæ are straight or inclined in order to fix those directions as in other animals in which the otolith could move." Breuer is constrained to the opinion that the otolith of the macula utriculi moves in the horizontal direction from behind and external toward internal and anterior, while the otolith of the macula sacculi is said to move in the direction from above and posterior toward below and anterior. In spite of our more perfected histologic technic, no one since Breuer has taken upon himself the trouble to control Breuer's statements. In a way which has not pro-

duced results hitherto, Ruijsch has attempted to solve this question of "Gleitrichtung." He has taken Roentgen photographs before and during the movement of fish heads imbedded in paraffin. The limit of error in the demonstration of the position of the otoliths amounts, however, to about 0.1 millimeter, and it is impossible that the otolith moves such a distance. It can be a question at most of only a hundredth part of a millimeter. It is, therefore, clear that this method is useless. In fact, Ruijsch could not determine a movement of the otoliths, which, nevertheless, demonstrates nothing against Breuer's assumption. Breuer is impelled to his hypothesis chiefly through the structure of the nerve end places in bony fishes, where the nerve end place is situated in the longitudinal groove of the otolith, so that the otolith, in Breuer's opinion, can be moved only in a longitudinal direction, but not transversely. Ruijsch cites, however, a work of Fryd, in which more exact determinations concerning the anatomic relations of the otoliths were made. From this work it follows that the groove of the otolith very frequently presents elevations and depressions which, according to Ruijsch, must make impossible a movement in the sense of Breuer. Moreover, he finds at the anterior and posterior ends of the longitudinal groove, detached lateral surfaces of the otolith by which it appears to be connected to the nerve end plate. This fact also speaks decidedly against the glide hypothesis (gleit-hypothese). I cannot see why a glide movement should be necessary for the otoliths. It is indeed completely sufficient for the excitation, if the otolith, as a result of its greater specific weight, exercises a pull upon the hairs of the hair cells, now to the one, now to the other side of the nerve end place, according to the direction of the nerve end place in space. It seems to me, therefore, that the question whether a gliding movement exists or not is still unanswered, and it lies within the province of the histologist to investigate this question still further.

Breuer and Mach have assumed that the otolith organs are destined solely for the sensing of position and for the perception of progressive accelerations. Bartels questions why they should not be excited also through turnings, since every turning of the head must indeed bring about a backward pressure of the specifically heavier otoliths opposite to the turning direction.

Let us imagine that the turning axis passes through the otolith itself, then the turning could produce solely a torsion of the otolith upon its nerve end place. Even if such a condition could in reality take place, the hairs as a whole would be stretched and the entire mass of hair cells would be excited. We must assume, in accordance with the experiments of Kubos, that the hair cells of the various sides of the end organs are antagonistic in their function. There would take place, therefore, upon simultaneous excitation of all hair cells a reciprocal inhibition. In spite of this, it is still questionable whether such a reaction takes place. It is conceivable that the antagonistic cell groups, as well as those in the semicircular canals, are unequally excitable. At any rate, it appears that the otolith organs are less adapted to excitation through turning than to excitation through gravity and progressive movement. Inasmuch as a progressive movement is combined with turning around an axis which does not pass through the otolith, the otolith organ must naturally be excited by turning movements. Also centrifugal force must influence the otolith. (Breuer and Kreidl.)

We have now to study the centrifugal reflexes and sensations from the otoliths. Mulder observed in guinea pigs a head reflex which consists of a backward movement of the head opposite to a straight line accelerated movement. By means of an apparatus, the kind of movement and the moment of the origin of the reflex were exactly measured. Mulder stated that the reflex time amounts to about 0.12 of a second. If the acceleration of the progressive movement lasts a shorter time than 0.12 of a second, therefore shorter than the time preceding the beginning of the reflex, then the acceleration speed must be extraordinarily great in order to produce any reflex at all. The most favorable condition obtains when the acceleration is taking place when the reflex begins. The smallest rate of rotation which Mulder found capable of producing a reflex amounts to five centimeters per second. In man there is no head reflex as in guinea pigs. In animals there are to be observed extraordinarily exact reflex movements and position of the eyes in varying directions, according to the position of the head in space. The dependence of these movements upon the otolith organs have been proven by a number of investigators. (Breuer, Lee and Kubos.) These eye movements

result when one changes from the upright head position to conform to the slow movement of nystagmus during turning, which is necessary to the attainment of the concerned head position. They fix in the new position of the head a part of the slow movement of the nystagmus which is elicited during turning. It follows that there must exist the same central connections for the nerve end places of the semicircular canals as for the otolith organs with the eye muscle nuclei. The equality would be still greater if gravity also acted upon the nerve end places of the semicircular canals. In mammals and birds one has not hitherto succeeded in irritating the nerve end places of the otolith organs singly. Unfortunately, Wittmaack says nothing in his turning investigations on animals concerning the persistent changes in position of the eyes upon varying positions of the head. This should be especially interesting because from these experiments of isolated tearing away of the otoliths and isolated loss of function of the otolith end organs, some conclusion might be drawn as to the influence of gravity upon the ampulla end organs.

In normal human beings the kind of reflex positions of the eyes seen in animals are not to be found. Only rolling of the eyes to the opposite side upon lateral inclination of the head causes a similar reflex, originating in all probability in the otolith end organs. (Breuer.) The movements of the eyes in man have been measured solely by subjective methods. (Hueck, Javal, Nagel, Breuer and Kreidl.) I have constructed an apparatus which permits the measuring of this movement in an objective manner. I have described this apparatus and the results of my measurements repeatedly, and believe, therefore, since I could add nothing at present, that it is unnecessary to describe it again in this place.

Breuer has observed in blind people eye movements analogous to the eye movements of animals. In the normal person the influence of voluntary innervation prevents these movements from becoming manifest. A vestibular tone of the eye muscles, which goes out chiefly from the otolith organs, may, however, also be present in man. Ewald believes that a tonus from the labyrinth goes out to the musculature of the whole body. Breuer believed that disturbances of equilibrium in man arising upon galvanization of the head depends upon a disturbance of this tonus. I was able to show that these disturbances

of equilibrium stand in relation to the rotatory nystagmus produced by the semicircular canal apparatus, and consequently there is no occasion to ascribe them to the otolith organs. I have demonstrated in man the influence of the otolith organs upon the innervation of the musculature of the extremities, concerning which something will later be said. The brief sensations arising from progressive movements probably owe their origin chiefly to the otolith organs. These sensations have not been exactly studied.

The knowledge of the position of the head probably depends in man upon muscle and joint sensations; for, as Alexander and I have found, the determination of the head position by bending of the head upon the trunk, and the determination of the head-body position upon lateral inclination of the entire body and head, are subject to the same mistakes in the normal as well as in the deaf-dumb with destroyed vestibular apparatus.

(d) The Vestibular Nerve and Its Connections.

The following statement holds both for the ampullar and the otolith nerves, since an isolated examination of these fibers has not yet been possible. Before its entrance into the medulla oblongata the vestibular nerve passes through the bipolar ganglion cells of the vestibular ganglion. According to Lenhossek, the central process of such a ganglion cell is frequently thicker than the peripheral process, which is shown by an increase in the number of the fibrils in the area proximal to the ganglion itself. Winkler calls attention to the fact that the ganglion cells of the vestibular ganglion are interspersed along the vestibular nerve, and that the vestibular ganglion, at least in canines, lies near to the medulla oblongata. This may be important, since destruction of the peripheral nerve end places of the semicircular canals brings about a condition similar to division of a peripheral nerve, and the degenerations arising after such operation are different (Marx, Trendelenburg), just as if the ganglion cells of the vestibular ganglion were themselves destroyed (Winkler).

Upon its entrance into the medulla oblongata, the vestibular nerve forms the ventral and proximal root of the nervus acusticus, which is found anterior to the corpus restiforme, between the latter and the descending root of the trigeminus.

According to Cajal, the root fibers of the vestibular nerve end in four nuclei:

1. In the dorsal or triangular or principal nucleus.
2. In the Deiters' nucleus.
3. In the Bechterew's nucleus.
4. In the descending vestibular nucleus.

Cajal supports his contention chiefly by Golgi preparations. Winkler asserts, on the contrary, as a result of Marchi preparations, that for the canines, at least, a part of the root fibers of the vestibular nerve seeks out the nuclei of the cochlear nerve, and that vice versa fibers from the cochlear nerve enter into the nuclei of the vestibular nerve. If we try to harmonize this well-supported view of Winkler with the facts of physiology, that would mean that in the semicircular canals and in the utricle and saccule tone impulses arise. Now, Kalischer has demonstrated, through his training method, that in dogs, even after apparently complete destruction of the cochlea, there remains behind a certain qualitative tone perception. This finding would harmonize with the views of Winkler, provided that in dogs this transition of vestibular fibers into the cochlear nuclei also takes place. In mankind the semicircular canals are certainly not excitable for tones. That is demonstrated by the numerous cases of complete deafness with excellent irritability of the semicircular canals. There is no explanation for the endings of the cochlear nerve in the nuclei of the vestibular nerve. It is not impossible that impulses of the cochlear nerve produce a certain muscle tonus, as this certainly occurs in man through rhythmic music.

One of the most important discoveries for which we are indebted to the Golgi method concerns the bifurcation of the root fibers of the vestibular nerve, as described by Kölliker. Kölliker asserts that the root fibers of the vestibular nerve divide behind the descending branch of the trigeminus. Cajal says (page 760, volume I) that each fiber of the vestibular nerve is divided in the form of a "Y," into two branches: into an ascending thinner branch, and into a descending thicker branch. The ascending branch gives off numerous collaterals to the apex of Deiters' nucleus, and to Bechterew's nucleus, and becomes then a constituent part of the vestibulocerebellar tract. (Volume I, page 764; volume II, page 141.) According to Cajal,

the fibers of this tract consist solely of the ascending branches of the vestibular nerve which enter through the nuclei of the cerebellum and end in the cortex of the cerebellum both in the right and left vermis, and also in both hemispheres. The descending branch gives off a great number of collaterals to the cells of Deiters' nucleus, to the triangular nucleus and to the descending vestibular nucleus, ending finally in the latter. The axones of the cells of these four nuclei form the socalled central tracts of the vestibular nerve. Cajal differentiates (volume I, page 773) here an uncrossed and a crossed tract. Both contain ascending and descending fibers. The fibers of the uncrossed tract descend for the most part to the medulla, but a few divide into an ascending and descending branch. The fibers of the crossed tract belong to the posterior longitudinal bundle and are for the least part ascending fibers, most of which divide into an ascending and descending branch. Cajal sets forth several physiologic observations (volume I, page 827), from which we abstract the following:

The ascending fibers in the posterior longitudinal bundle serve for the connection of the nuclei of the vestibular nerve with the eye muscle nuclei; the descending branch of the connection goes to the motor anterior horn cells of the spinal cord for the innervation of the head and trunk. The course of the impulse in the central tracts of the vestibular nerve Cajal gives as an example of what he calls "avalanche-like enlargements" for the conduction of nerve energy. The impulse which is taken up by a group of hair cells passes chiefly into a cell of the vestibular ganglion. Through the tremendous number of collaterals of the root fibers of the vestibular nerve, the impulse becomes, however, distributed to an extraordinary number of nerve cells, so that only a few cells of the sense organ can exert any influence upon the remote regions of the spinal cord and brain.

The statement of Cajal is not free from inexactness, and holds good only for animals. It would be, however, unreasonable should we attempt corrections because of the statements of other authors. It appears to me to be more correct, through data furnished by physiology and pathology, to point out the necessity of certain connections according to which in the above mentioned statement the main outlines at any rate of the anatomic relations are given.

(*e*) *The Centrifugal Reflexes and Sensations Elicited from the Vestibular Apparatus.*

If we set up in a normal man a strong irritation in the semicircular canals, we may observe a series of symptoms which in quite similar fashion are to be produced in animals. These symptoms are:

1. Nystagmus of the eyes and head.
2. Reaction movements—
   (a) Of the extremities.
   (b) Of the body.
3. Turning sensations.
4. Nausea and vomiting (sea-sickness).

If an endolymph movement takes place in the right horizontal canal toward the ampulla, then there arises a nystagmus of both eyes toward the right. That each endolymph movement calls forth a nystagmus in the plane of its movement and contrary to its direction, I probably need not dilate upon at this point. This has already been sufficiently described. I should like here to go into the origin of nystagmus, since we, in recent times especially, have learned some new and important things through the splendid investigations of Bartels.

Vestibular nystagmus toward the right begins, as is known, with a slow movement toward the left, which is followed by a quick movement toward the right. Hogyes had already stated that if the muscles of the eyeball were cut through, except the rectus externus or internus, this one muscle suffices to produce typical vestibular nystagmus. Bartels has proved this observation with absolute certainty by separating the muscles from the eyeball and recording their twitchings directly. He was able to show that both the rectus internus and rectus externus of the right and left eyes can produce nystagmus respectively toward the right and toward the left. Let us observe the behavior of the right external rectus muscle in nystagmus toward the right. The slow movement of the right eye toward the left can only be produced through a relaxation of the right rectus externus, and in fact such an active relaxation represents the first phase of the movement. Then follows the quick movement in the form of a rapid contraction. The rectus internus of the right eye shows exactly the reverse. The slow

movement toward the left depends upon a slow contraction of the rectus internus, but a quick relaxation of this takes place during the quick movement of the nystagmus, etc. Again, the relaxation of the rectus externus and internus in the left eye is contrary to that of the right. We may determine with certainty that a contraction of the rectus internus always corresponds to a relaxation of the rectus externus, and vice versa. Especially important appears the fact that the relaxation of the eye muscles also takes place if the eyes were previously in the resting position. The relaxation in nystagmus is, therefore, merely an increase of the normally existing rest tonus. The variation between contraction and relaxation produces the nystagmus which each muscle is capable of describing for itself. If we now turn back to horizontal nystagmus toward the right, we may conclude from the above mentioned data that the displacement of the cupola in the right horizontal canal toward the utriculus brings about the following nerve reactions upon the eye muscles:

1. Slow relaxation of the right rectus externus.
2. Slow contraction of the right rectus internus.
3. Slow relaxation of the left rectus internus.
4. Slow contraction of the left rectus externus.

A simultaneous contraction of the one and relaxation of the other muscle one might think could be of peripheral origin, if the supposed rest tonus arising from the sense cells of the ampulla should exist. It is also to be concluded from the described eye movements that the sense cells of the crest on the canal side of the horizontal canal stand in connection with the right rectus internus muscle and left rectus externus muscle, while the sense cells of the utricular side are connected with the right rectus externus muscle and left rectus externus. The stretching of the hair cells on the canal side could then bring about the contraction of the right rectus internus muscle and left rectus externus, while the relaxation of the hairs on the canal side would have as a result relaxation of the rest tonus, and with it a relaxation of the right rectus externus muscle and left rectus internus. This would be an example of a peripherally excited inhibition of innervation.

If the rest tonus does not exist, however, then the relaxation of the hair processes probably cannot bring about inhibition,

and the connection between contraction of the one and relaxation of the other muscle must be purely of central origin. At any rate, we must assume a central connection, for the centrally produced nystagmus shows the same character as that of peripheral origin. I have demonstrated that only a slow movement of the nystagmus is produced in the labyrinth while the quick movement is of central origin. Bartels assumes that the quick movement is produced in the cortex of the cerebrum through irritations which arise in the eye muscles during the eye movement. These irritations are said to pass through the trigeminus to the cerebral cortex, and from there to the eye muscle nuclei. These assumptions are, however, certainly to be looked upon as incorrect.

First.—Tozer and Sherrington have demonstrated that the tendinous bodies present in the eye muscle (sensitive nerve endings) are supplied not by the trigeminus but by the eye muscle nerves themselves.

Second.—Animals without a cerebrum show completely normal eye nystagmus, as Flourens and Ewald have already demonstrated, and as I myself have recently proved in a dog operated upon by Rothmann, in which the entire cerebrum was lacking. Therefore, the quick movement cannot be produced in the cerebral cortex. The experiments of Bartels, in which both rectus externus and internus described nystagmus in both directions, do not prove that in central third nerve paralysis the unparalyzed rectus externus must produce nystagmus toward both sides. Bartels has not proved that upon division of the oculomotor nerve the rectus externus produces nystagmus toward both sides, and still less has he shown that this occurs in oculomotor paralysis of central origin. It is credible that contraction and relaxation of agonist and antagonist are connected in the same central manner, and that without contraction of the agonist, relaxation of the antagonist does not arise. This explains why Bartels and I in several cases where one had to assume a central oculomotor paralysis, could not produce nystagmus toward the side of the paralysis. Cases with abducens paralysis are not adapted to the solution of this question, since here still other muscles may produce the nystagmus toward the outer canthus.

I have found myself in error in regard to this point in my first publication, where I assumed that through absence of

a supranuclear paralysis, nystagmus must always be present in every direction. According to Sherrington, if the centripetal impulses from the paralyzed muscle during relaxation play an important role for the origin of the quick component of the nystagmus, then nystagmus toward the paralyzed side must be lacking in peripheric oculomotor paralysis. If, however, the quick movement is of central origin, then there must be produced in pure peripheric oculomotor paralysis a nystagmus toward the diseased side, whereas this nystagmus is lacking in paralysis of the oculomotor nucleus. Between nuclear paralysis and supranuclear paralysis the only point of difference seems to be the addition of mobility in vestibular irritations toward the side of the paralysis, when one produces a nystagmus in the direction of the rectus externus. To clear up this point further investigations must be carried out. My description of the vestibular eye movements appears to me not to be overthrown by the discoveries of Bartels and Sherrington, but only expanded and deepened.

As concerns the relations of the nervus vestibularis to the individual eye muscle nuclei, it may be shown without further ado that nystagmus toward every direction in space may be produced in the sound labyrinth when the opposite labyrinth is destroyed. But the nystagmus toward the destroyed side is usually very trifling in intensity and direction. Naturally, caloric nystagmus may be produced in the sound labyrinth toward the right and left. From these facts came the hypothesis enunciated by Bartels and accepted by Ewald, that caloric nystagmus is the result of paralysis of the vestibular nerve by cold.* If it happens in rare cases that after unilateral destruction of the labyrinth the other labyrinth also becomes negative for the caloric irritation, this does not speak, as Bartels thinks, against my caloric theory, but depends only upon changes in the central excitability, which indeed is almost always diminished after unilateral labyrinthine destruction.

The fact that nystagmus in any desired direction may be produced from the one labyrinth, demonstrates that every vestibular nerve must possess connections with all the eye muscle nuclei of both sides. Since nystagmus to the sound

*The hypothesis of Bartels and Ewald is naturally also made untenable through the influence of the change of the head position upon the direction of the caloric nystagmus.

side is stronger both in animals and man, the crossed connections must possess the greater function. Wallenberg has found that in the pigeon the uncrossed connections are the more powerful. Here lies another discrepancy between anatomy and physiology which must be explained.

We owe to Flourens the first communication concerning the influence of the canal apparatus upon the labyrinth musculature, who exactly described the disturbances of movement of pigeons in the plane of the divided canal. Ewald added something new in his statement of the centripetal muscle tonus elicited from the vestibular apparatus. Ewald believes that definite muscle groups are in relation with each labyrinth. Lange, a pupil of Ewald, thinks he has demonstrated in pigeons that the cerebellum has no influence upon the symptoms arising after division of the semicircular canal. The head movements simply begin earlier. Beyer and Lewandowsky, on the contrary, find great similarity between the symptoms after destruction of a vestibular apparatus and after cerebellar extirpation. But none of these authors has investigated how the turning is related to observed normal reaction movements after cerebellar extirpation. The influence of the position of the reaction movements after turning observable in normal individuals behave after cerebellar extirpation. The influence of the position of the head upon the direction of the reaction movements in animals which I discovered in man, has also not yet been investigated. In the following paragraph I will give a presentation of the methods of testing the reaction movements worked out by me, and will also go into the anatomy which it is here necessary to understand.

If we seat a normal person upon a chair and with the head in an upright position produce horizontal nystagmus toward the right through turning or irrigation tests, and ask the subject to stretch forth his right arm and hold it absolutely still, then we will observe that while the subject believes he is carrying out our request, his arm is in reality deviating slowly toward the left. This slow deviation of the arm appears in its rate of movement to be completely analogous to the slow movement of nystagmus, only the arm fails to perform the quick movement in the opposite direction, that is, toward the right. Now, how can we explain this deviation? The tested person has the task of holding his arm at rest. He does this so

long as he continues to send forth the same cortical impulse to his arm; but, finally, in spite of the persistence of the cortical impulse, he moves his arm slowly toward the left. This movement originates in the cerebellum, and is secondarily conditioned by the vestibular apparatus. If the subject notices, as a result of conscious muscle and joint sensations, that his arm moves, he tries to prevent this voluntarily, and one then sometimes sees nystagmus-like twitchings of the arm, viz., a slow movement toward the left and a quick correcting movement toward the right.

The less educated a person is, the less is he capable of guarding his muscle and joint sensations, and hence the greater this slow movement becomes manifest. In children I have repeatedly observed a deviation of nearly 180 degrees. In educated persons the slight mental haziness which arises through dizziness associated with the nystagmus, may call forth large reactions; but as a rule the reactions in such subjects are much slighter. For clinical purposes another kind of test may be applied. It is the socalled "pointing test" introduced by me. This test has the advantage that one can try out all joints of the extremities, while the stretching forth of a limb works only for the shoulder and hip joints. The pointing test is thus carried out: The subject closes his eyes and touches the pad of my supinated index finger; then rests his arm upon his knee and raises his arm again until his finger touches mine. If nystagmus exists toward the right, the subject does not touch my finger but points beyond it toward the left.

Upon carrying out this test it is absolutely necessary that the investigated person knows nothing of the manner in which he performs it; for this reaction is partly of cortical origin and therefore is influenced by suggestion and autosuggestion. One must avoid such an occurrence by keeping the subject in ignorance of his mistakes. Whenever, therefore, a mistake is made, the investigator moves his own finger so that it shall meet the finger of the subject, who will then not realize that he has changed the position of his arm. I can also test by this method isolated movements in the wrist joint, and this latter is especially adapted for expounding the theory of these movements. I will therefore use this test as the basis of the following discussion.

For trying out the reaction movements in the wrist joint,

the subject places his right forearm over the back of a chair and I hold it firmly with my left hand. Now the subject extends his wrist joint, and with outstretched index finger (the remaining fingers being closed in the palm of the hand) is instructed to touch my right index finger, held in the same manner along its entire length, then he bends his wrist joint as far as possible downwards. The joint is again extended, and the process repeated as often as one wills. In this test the entire movement is confined only to the wrist joint. If horizontal nystagmus exists toward the right, then the subject with head upright points past my finger toward the left. It is to be noted that many normal intelligent persons show no deviation or only slight deviation during the nystagmus, which evidently depends upon the great exactness of the muscle and joint sensations of this joint. In ignorant persons, however, one finds the deviation very frequent. But if the subject let the hand hang loosely over the back of the chair instead of carrying out the movement, one then sees not the slightest deviation. The origin of the reaction movements is therefore dependent upon voluntary innervation. This is a fundamental fact which belongs to the vestibular reaction movements in man; in animals, where cortical function is slighter, it appears to be quite otherwise.

The described deviation toward the left takes place by every given position of the arm. I can pronate the arm and place the flexor surface of the lower arm upon the back of a chair. I can also supinate the arm and fix the dorsal surface of the lower arm upon the back of a chair. Then, in this position, there arises during nystagmus toward the right, pointing deviation toward the left. Also, by every other chosen position of the lower arm, if motion is produced in the wrist joint in a vertical direction from below upward, or vice versa, deviation toward the left always arises. Let us make clear the muscle action which causes deviation toward the left when the lower arm lies pronated across the back of a chair. The extension of the wrist joint is produced by the extensor carpi radialis and ulnaris, flexion by the flexor carpi radialis and ulnaris. If upon pronation of the forearm a deviation to the left shall occur, the radial muscles, viz., in the case of dorsal flexion, the extensor carpi radialis, must receive additional innervation. In other words, the persisting cortical innervation must be re-

inforced by a subconscious innervation originating from the cerebellum. At the same time the cortical innervation of the extensor carpi ulnaris is diminished. During flexion, on the contrary, the cerebellar innervation of the flexor carpi radialis is increased while that of the flexor carpi ulnaris is diminished. If we now supinate the forearm and place its dorsal surface upon the chair back, then with the resulting deviation to the left during flexion of the wrist the cerebellar innervation of the flexor carpi ulnaris is increased, that of the flexor carpi radialis is decreased; while with dorsal flexion the extensor carpi ulnaris is innervated, and the extensor carpi radialis is inhibited. Therefore, with every change in position of the hand there is a corresponding change of nervous impulse. What does this signify when translated into the language of brain anatomy? From the canal apparatus the same irritations must naturally always go out, whether the forearm is pronated or supinated. The innervation of the muscles of the lower arm is, however, a quite different one according to the position of this member. The position of the extremities is under voluntary control and is governed by the cerebral cortex. Therefore, the innervation of the cerebral cortex must determine which muscles are influenced by the vestibular irritation, whether the radial or the ulnar. The cortex of the cerebellum is the place where this influence can and does take place. We know that from the motor area of the cerebrum the pyramidal tract conducts motor impulses to the spinal cord. In the pons, however, as Cajal has demonstrated, every pyramidal fiber gives off a collateral to the pons nuclei, and from there a new neuron passes out through the peduncle into the opposite cerebellar hemisphere and here gives off collaterals, which, as Cajal deduces from the embryologic development, and as I must assume upon clinical grounds, ends as a climbing fiber (fibre grimpante) around a Purkinje cell. We have here, therefore, one way in which the voluntary innervation to several places of the cerebellar cortex is effected in quite similar fashion. In the cerebellar cortex, however, the cortical and vestibular innervation meet each other; for Cajal has demonstrated, as already mentioned, that every fiber of the nervus vestibularis ends in the cerebellar cortex. Since each vestibular impulse is conducted by a great number of fibers in the same manner, and each of these fibers gives off numerous collaterals in the cere-

bellum (Cajal), then it is not so difficult to imagine that the entire cerebellum is subjected to the influence of a fixed vestibular irritation. But to this propagation of a centripetal impulse, the special structure of the cerebellar cortex also contributes, into which thing I should like here to go, in order to mention the principal facts of cerebellar histology. We differentiate in the cerebellar cortex three layers:

First.—The layer of parallel fibers.
Second.—The layer of Purkinje cells.
Third.—The granular layer of cells.

Let us begin with the second layer. The Purkinje cells are, as is known, extraordinarily large ganglion cells which send upward into the layer of parallel fibers a large tree of dendrites, while they send their axis cylinders downward, to end, according to Cajal, in the nuclei of the cerebellum. The Purkinje cells are not strewn about heterogeneously in the cerebellar cortex, but their arrangement is seen to be remarkably regular. They stand palisade fashion in the sagittal plane behind each other, their dendrites are flattened from right to left and hence their greatest degree of expansion lies in the anteroposterior direction. Beneath the Purkinje cells lie the granular cells. These are present in very great number. Obersteiner reckons about 60 granular cells to one Purkinje cell. The small ganglion cells, each one of which possesses five to six dendrites, after a short course, end in a peculiar manner in the glomeruli of the cerebellum. These glomeruli are probably nothing but nodal points in which several nerve fibers meet and join with each other. We shall learn something more about these nerve fibers later. The axis cylinders of the granular cell have a regular course. Every granular cell sends out its axis cylinders upwards into the parallel fiber layer of the cerebellum. Here, this axis cylinder divides into two branches and the two run in a frontal plane toward the right and left for some distance, passing through the dendrites of the Purkinje cells. The Purkinje cells may be described as telegraph poles and the axis cylinders of the granular cells as telegraph wires. A difference, however, exists in that here the telegraph poles do not merely hold the wires but also receive the current and carry it farther on. If the vestibular impulse is imparted to the granular cell, and this is nearly certain, then this arrangement

can be understood, since one and the same vestibular irritation can influence a great number of Purkinje cells. It is very probable that this irritation works strongest upon the nearest lying and always weaker upon the farther lying Purkinje cells, as we shall see later. In contradistinction to the vestibular irritation which is not individually but collectively distributed, the voluntary irritation which travels along the climbing fibers leading to the Purkinje cells has a purely individual character. We have already heard that the axones of the Purkinje cells end in the central cerebellar nuclei. From here originate new neurones which betake themselves into the spinal cord in a complicated manner. From the nucleus of the hemispheres to the corpus dentatum passes the brachium conjunctivum, crossing, as is known, to the red nucleus of the opposite side, and from here originates the rubrospinal path of Monakow, which crosses and ends around the anterior horn cells of the spinal cord. In this manner each cerebellar hemisphere is in connection with the anterior horn cells of the same side, and with the extremities of the same side. We can now understand how in the cerebellar cortex the voluntary innervation works together with the vestibular innervation and the new formed irritation is carried over to the same anterior horn cells to which the voluntary irritation also passes directly by means of the pyramidal tract. In order to penetrate further into the mazes of this complicated mechanism, we must study further facts. We have heretofore merely taken into account the horizontal nystagmus toward the right upon upright head position which produced the pointing reaction toward the left. We can, however, produce the pointing reaction toward the left during any given nystagmus, if we bring the head into a corresponding position. The nystagmus only must be directed in a horizontal plane toward the right. In practice the thing is very simple. If we turn a man toward the left with head upright, then he experiences upon stopping horizontal nystagmus toward the right. Thereupon he points toward the left. I will now change the position of the head. If I turn the patient toward the left and he holds his head in the same position upon stopping in which it was placed during the turning, then the after nystagmus always moves in a horizontal plane toward the right, and the pointing reaction, therefore, takes place toward the left. The pointing reaction toward the

left is, therefore, a function of two variables, one of which is the canal irritation and the other the head position irritation. To each value of the canal irritation there is a corresponding value of the head position irritation in order to produce together a pointing reaction in a fixed direction. The coalition of these two irritations probably takes place in the glomeruli of the cerebellar cortex, so that the parallel fibers take up the new formed irritation and impart it to the Purkinje cells. It is also to be taken for granted that if the pointing reaction in varying combinations of these irritations always deviates toward the left, it must be always the same Purkinje cells which here come into play, so that all possible vestibular irritations must be able to affect each Purkinje cell. Moreover, the arrangement of the parallel fibers seems here to be of advantage, since the concerned irritation can be carried to various points of the cerebellar cortex through mediation of the parallel fibers and can be conducted to remote Purkinje cells. Here it is in place, perhaps, to state that the Purkinje cells are not merely connected in the frontal plane through the parallel fibers, but that they are also in communication in the sagittal plane. This takes place through mediation of the socalled basket cells. There are in the parallel fiber layer large ganglion cells which send out anteriorly and posteriorly very long axis cylinder processes from which a collateral is given off to every Purkinje cell, which surrounds this cell with a plexus of fibers. The basket cells appear to be intended to carry over a given vestibular irritation to a frontal row of Purkinje cells and to carry this to another frontal row of cells, and so on.

From the statements already presented we may draw several conclusions as to the pathology. If those Purkinje cells of the right cerebellar hemisphere which produce the movement toward the left during nystagmus toward the right are destroyed, then we shall see no movement in the right wrist joint toward the left during normally produced nystagmus toward the right, and this is quite independent of what kind of nystagmus is produced during turning toward the left, if only the nystagmus always strikes toward the right. In diseases of the fibers, however, it may happen that for one form of nystagmus the reaction fails toward the left, but is retained for another form. What happens, however, if we change the arm from its prone position into the supine position? In this case the

same Purkinje cells can no longer functionate, for the Purkinje cells functionating with the arm in a prone position are in relation with the flexor and extensor carpi radialis, while upon supine position of the arm, the flexor and extensor carpi ulnaris must be innervated for the movement toward the left.

It appears, a priori, quite possible that we can have merely a paralysis of the movement toward the left with the arm in a prone position, while with the arm in a supine position a reaction may still be present. As a matter of fact, I have observed an entire series of cases in which this relation was present. In a cerebellar abscess operated upon by me and healed, the reaction movement toward the left was lacking also upon supine position of the forearm. Since in this case the reaction movements in all other joints were normal, and the abscess had been very small, it is to be assumed that the Purkinje cells, which are in relation to the flexor and extensor carpi ulnaris, do not lie far from those which control the flexor and extensor carpi radialis. But there is a further question to be considered. If I hold the arm pronated, then during the nystagmus toward the right the flexor and extensor carpi radialis are innervated and bring about the pointing reaction toward the left. If the center for this movement in the cerebellum is paralyzed, then the pointing reaction toward the left is wanting during nystagmus toward the right. Now what happens if I produce a nystagmus toward the left with the arm in a supine position and test the reaction movements? In the normal person the pointing reaction should take place toward the right, since the flexor and extensor carpi radialis are in turn innervated from the cerebellum. But we have heard that the center for these two muscles is paralyzed, at least in so far as the same are concerned in the movement toward the left with the arm in pronated position. If there is only one center in the cerebellum, however, for these two muscles, then they must be paralyzed both for the movement toward the right with arm in pronated position, and for the movement toward the left with arm in supinated position, but that is not the case. The same muscles which produce no left movement in the pronated arm upon nystagmus toward the right, produce a marked movement toward the right with the arm supinated, when the nystagmus is directed toward the left. From that fact one may conclude that these two muscles must have at least two representations

in the cerebellar cortex: there must be one center for movement toward the right and one for movement toward the left, in which all the arm muscles are represented.

Now let us consider the collaterals of the pons fibers which conduct voluntary impulses to various places in the cerebellum. These varied places in the cerebellar cortex must receive the same voluntary innervation, since a given muscle is represented in many places of the cerebellar cortex. Now, how many centers of control may a muscle possess? In answer to this question I have no experimental or pathologic material, but upon theoretical grounds and from clinical cases I have drawn the conclusion that each muscle must be represented at least four times. I have observed cases in which the reaction both toward the right and left were lacking, and others in which the reactions toward the right and left were normal and a reaction from above or below was gone. From this we may conclude a fourfold representation for the musculature. I have still to add that pathologic cases have occurred in which various joints of the extremities seemed to possess special areas in the cerebellar cortex. There are cases in which the reaction movement in a given direction is paralyzed in the wrist joint but which in the shoulder joint is normal, and again cases in which in the hip joint and shoulder joint the movement in a given direction is lacking, and still others in which the movement is wanting only in the shoulder joint but is present in the hip joint. We may conclude, therefore, the following: In the cerebellar cortex there is a representation for controlling the musculature of the joints and for directions of movement. Each direction of movement is represented only once. Each joint and each muscle, however, is represented at least four times in one hemisphere.

From the fact that the pointing reaction in a given direction is a function of canal irritation and head position irritation, it necessarily follows that if the one irritation remains constant the other is changed and the pointing reaction must also be changed. It appears, therefore, quite clear that if by upright position of the head horizontal nystagmus is produced toward the right, the pointing reaction takes place in a horizontal direction toward the left; if we produce vertical nystagmus upward, the pointing reaction upon upright position of the head in the sagittal plane must take place downwards. If we

produce rotatory nystagmus toward the left, the pointing reaction must appear in the frontal plane with the right arm directed downwards and with the left arm directed upwards. Just as we can change the canal irritation by persistent position of the head, so we may also change the head position during a persistent canal irritation and with it change the pointing reaction. If we have a horizontal nystagmus toward the right, and incline the head 90 degrees toward the left shoulder, then the pointing reaction takes place downwards; if we incline the head 90 degrees toward the right shoulder, then the pointing reaction takes place upwards. If rotatory nystagmus exists toward the left, then by forward inclination of the head to 90 degrees the pointing reaction takes place in the horizontal plane toward the right, and by backward inclination to 90 degrees the pointing reaction takes place in the horizontal plane toward the left. In rotatory nystagmus toward the left, if we turn the head 90 degrees toward the left, then the subject points with both arms downwards in the sagittal plane in moving the arms from right or left; and if we turn the head 90 degrees toward the right, then the pointing reaction of both arms takes place in the sagittal plane in an upward direction. Every combination of the two irritations enables one to calculate the kind of pointing reaction.

These investigations have also led to new knowledge concerning the function of the otolith organs. Previously the question was whether the head position irritation could be attributed to the sensations arising in the head and neck joints and muscles, or also to the impulse in the otolith organs. If we bring about a change of the pointing reaction in rotatory nystagmus toward the left by turning the head around the vertical axis toward the right and left, then it cannot be a question of change of impulse in the otolith organs, for gravity works quite the same in all these positions. In this case the impulses from the head joints and neck joints and muscles must give off head position irritations and bring about a change of innervation. The path which conducts these sensations to the cerebellum is most probably the tract of Gowers (tractus spinocerebellaris ventralis). If the head is, however, inclined upon the shoulder, either forward or backward, then there must arise also sensations in the head and neck joints, but the nerve end places of the otolith organs receive likewise a changed impulse. Here,

therefore, a combined function may take place. I have succeeded in demonstrating that the otolith organs are also able to bring about changes of innervation. If, for example, I place a patient with horizontal nystagmus toward the left upon his back and ask him to perform the pointing reaction with the right wrist joint, then he points toward the right. If I now turn him while the arm rests on the back of a chair, so that he lies on his left side, then the head does not change its position opposite to the body, but the otolith organs receive a changed innervation. Moreover, in the side position a total change of the innervation of the wrist joint takes place. This demonstrates the influence of the otoliths on the change of vestibular tonus.

The reaction movements of the body are thoroughly discussed in the clinical part of this essay, and therefore I need not mention them now.

From what we have already described, one may conclude that the cerebellum is merely the central organ of the vestibular nerve. In fact, the musculature for direction of movement is controlled in the cerebellum and each of these centers is responsible to the vestibular apparatus; nevertheless, the vestibular apparatus plays only a subordinate rôle in man in determining physiologic cerebellar function. We must not forget that the vestibular fibers which enter the cerebellum make up only a small part of the centripetal path. The major part is supplied from the spinocerebellar tracts and the olivary tract in the corpus restiforme. The spinocerebellar fibers, according to common opinion, conduct muscle and joint sensations, "proprioceptive sensibility," after Sherrington.

According to the view of most authors who have worked with Marchi degeneration specimens, the spinocerebellar tracts end chiefly in the worm of the cerebellum, but the olivary fibers which pass, according to Cajal, Stewart and Holmes, from the olives to the cerebellar cortex, go solely to the opposite hemisphere. Moreover, the cortex of the hemispheres needs "proprioceptive sensibility" to be efficient. If, therefore, the spinocerebellar tracts do not enter into the hemispheres, we should have to assume that the olivary fibers conduct such proprioceptive impulses for the extremities which are brought to them in the anterolateral bundle of the spinal cord.

The chief task of the cerebellum may be to tone up and increase the efficiency of the musculature by means of the socalled proprioceptive reflexes. The vestibular apparatus also contributes to this tonus as well as the muscle and joint sensations. The function of the vestibular apparatus consists also, in my opinion, in a toning up of the various centers of the cerebellum, which takes place in the resting condition. The tonicity produced from the peripheral vestibular apparatus is not very essential, at least in man, for there are normal individuals in whom the strongest vestibular irritation only produces slight reaction movements and who are completely normal with respect to adroitness and equilibration. After unilateral labyrinth destruction, there is usually a very marked diminution of the vestibular excitability of the cerebellum without permanent awkwardness or disturbances of equilibrium or diminution of the muscle tonus becoming manifest. It has become possible, as I believe, to diagnosticate central diseases in the cerebellum by means of vestibular tests. Nevertheless, I see no reason to alter my opinion that the vestibular apparatus plays only a subordinate role for the maintenance of equilibrium and for muscle tonus, and on the whole represents a sense and reflex apparatus in the process of retrograde development. I have explained the grounds for this quite exactly at Basel and at the Psychological Congress in Innsbruck and could add nothing new to the same at this time.

Few authors have concerned themselves with the turning sensations since the work of Mach and Breuer. The observations of von Stein have more clinical interest. Important psychologic and physiologic conclusions have been determined by two workers in the school of Zwardemaaker, namely, van Rossem and Mulder. These investigations were carried out with an extraordinary amount of material, which is so absolutely necessary for the exact solution of the questions at hand. Both authors worked with a very exactly constructed electrically driven turning disk, the revolutions of which were recorded. The most important facts which van Rossem established are the following: In a very great number of investigations in which van Rossem himself served as subject, he determined the minimum of rapidity in which a turning sensation was just perceptible. In a series of investigations the

turning disk was set in motion at the same rate of rotation. If the subject experienced simply a turning and nothing more, he had to change the position of his head and to notice whether a turning sensation again appeared. It was found that the turning disk had to be rotated at a speed of 1° 30″ to the second to produce the sensation of turning. A second series of investigations, which made possible more exact results, was carried out. In these investigations the turning disk was set in motion at a certain rate of speed. The subject had to pay attention whether the turning sensation arose or not. Here the average minimum rate was 1° 36″. Exact recording and subsequent mathematical calculation showed that the final speed of 1° 36″ was reached in 1-45th of a second. From that it is shown that the acceleration must amount to 80° per second if it is to produce a turning sensation. How great the speed is in our clinical tests has not been exactly recorded up to the present time.

Van Rossem carried out further investigations concerning the perceptible minimum in various positions of the head, and found that an irritation of the vertical canals with the head bent backwards produced a turning sensation more easily than an irritation of the horizontal canals. Van Rossem laid this fact to the greater proportion of cupolæ in the vertical canals. I believe, however, that here central conditions play a rôle.

A great number of various attempts have been made to measure the reaction time of the turning sensation. An average of 0.8 of a second was found, which is a considerably greater time than is necessary in most other sense organs. Van Rossem believes that here peripheral conditions are the cause, an assumption with which I cannot agree.

A number of investigations are concerned with the measurement of the duration of the turning sensation. In two subjects van Rossem determined very different but harmonizing values. He believes that peripheral conditions are able to explain these things. I believe I have shown, however, that the center here plays the chief rôle.

Unpleasant feelings were manifest in all the subjects experimented upon, in that they experienced a sensation of turning without turning actually having taken place. Van Rossem has avoided these sources of error through skillful manipula-

tion of his apparatus. He has proved in five persons that the reaction time and the level of the sensation tally in every essential with each other. Mulder has carried van Rossem's work further. By means of sensitive apparatus he sought to determine the reaction time of a nystagmus produced through turning. In this he did not succeed, for his apparatus was not capable of registering the speed of this reflex. Certainly, the recation time amounts to less than 6/100ths of a second; therefore, less than the 1/10th part of a reaction time which is necessary for the sensation itself (8/10ths of a second). This demonstrates that the long reaction time of the sensation is due solely to central causes, and since it cannot be due to nerve conduction, then it must be due to the action of conveyed impulses in the centers. The determinations of the threshold of sensation are very interesting. Van Rossem had found, as above mentioned, that if the final speed of 1° 36″ in 1/45th of a second was reached, the sensation took place. This corresponds to a speed of 80° per second, which, however, only takes place in 1/45th of a second. Mulder perceived that the short duration of the speed impulse is the reason why the degree of the acceleration must take on such a high value. He produced a uniform acceleration through at least one second, therefore, about the necessary time for a reaction, and found that the sensation caused by uniform turning arises if the acceleration amounts to at least 2° per second and if the uniformly increased speed continues at least one second. If the speed of the equally accelerated turning amounts to at least 3°, then one experiences an accelerated turning.

It is especially noteworthy that, as mathematical calculation showed, the physical work performed by the minimum irritation which just produces a sensation is quite the same in the investigations both of van Rossem and Mulder; although the methods for the determination of the threshold of irritation are completely different.

In a series of investigations the threshold of difference was determined between two consecutive turning sensations which were produced through varying rapidity of rotation. The subject had to say whether the first or the second turning appeared quicker. Both speeds were reached in approximately the same time. It was obvious that Weber's law also holds good here. The slower the turning, the greater the differ-

ences had to be in order that another variation be noticed. In general, two irritations which were differentiated at about 1/10th of the irritation strength, were still perceived separately.

The investigations concerning blending and reciprocal suppression of turning sensations are interesting. If one revolves the turning disk at a speed of 24 degrees per second and suddenly interrupts the turning so that the pause lasts just as long as the previous turning, then one obtains varying sensations, according to the number of interruptions in a given time. If the time of the periods of the turning and pause amounts to 9/10ths of a second, then one experiences first the actual turning, then the pause, and after that the reversed apparent turning. If the time of the periods amounts to 7/10ths of a second, then one feels he is being turned now in the turning direction, now contrary to the same without perceiving the pause. There takes place in these shorter interrupted intervals a coalition of the two sensations, the sensation of real turning and that of apparent turning. If the time of the periods amounts, however, to 4/10ths of a second, then one usually experiences no turning, but believes he is standing still during the entire experiment.

To explain this Mulder concludes that the turning irritation works too short a time to produce a sensation. His explanation of this phenomenon consists in that as a result of the long reaction time (8/10ths of a second), the irritations cease before they can reach consciousness. I believe this phenomenon shows that the concerned excitations influence one another in a subcortical center, perhaps in the cerebellum. This center needs for the production of the excitation a fairly long time before it carries the irritation to the cortex of the cerebrum, therefore the long reaction time. If, now, an opposed irritation enters during the persistence of the first, then the two combat and annihilate each other before the cortex of the cerebrum receives any news of this fight in the subcortical region.

Mulder determined further the number of periods for different speeds necessary to produce the phenomena of apparent rest, and found that shorter periods belong to greater speeds, therefore to stronger irritations. From a great number of observations carried out by special methods, Mulder

then drew the curve of perception of turning which shows a quick rise, a maximum, and a slow decline.

In the discussion of seasickness in the physiologic section, it is set forth that here we do not have to do with an actual sickness; but with an extensive physiologic experiment out of bounds. The nausea associated with nystagmus is to be explained through the close relationship of the vestibular nucleus to the vagus nucleus. This varies in different individuals. There are normal persons who become severely nauseated by turning, but they are the exceptions. On the contrary, neurasthenic persons show the most severe nausea. Horizontal nystagmus causes the least disturbance. Strong rotatory and vertical nystagmus can, on the contrary, call forth nausea in many normal strong-nerved persons. Frequently, one or two turnings (each turning consisting of ten revolutions with sudden stop) brings about no nausea. If one repeats the turning oftener, then nausea may arise. Head movements produce a distinct influence upon the origin of nausea is effected during the turning or after stopping turning while the after-nystagmus lasts. We have here an analogy with seasickness in which nausea usually arises after the ship has been swaying for some time, and because of very irregular ship motions. Like neurasthenics, accident cases with concussion of the brain are also very much disposed to nausea. Further, multiple sclerosis appears to favor the production of nausea. Directly opposed to multiple sclerosis are those patients with cerebellar tumors, in whom nausea is completely absent during experimental irritation of the vestibular apparatus.

Moreover, nausea may be produced from the otolith organs, as experiences in an elevator and upon toboggan slides prove. Wojatschek has demonstrated in a readable work that nausea in seasickness depends, for the most part, upon the otolith organs. I proved that this nausea may be varied in different positions of the head. If one inclines the head 90 degrees forward or backward, thus changing an up-and-down motion into a movement forward and backward, then the nausea is in many persons more trifling than if the head is held upright. There are also persons who cannot bear to ride backward, and those may not be protected against nausea due to vertical movements by forward inclination of the head. During a

sea voyage in which the motions of the ship took place in a direction almost vertical, I proved myself with complete certainty that the position of the head has the greatest influence upon these sensations. I had already determined upon myself that quick up-and-down movements in an elevator with the head in an upright position called forth slight discomfort, but that forward inclination of the head to ninety degrees completely did away with this. To be sure, uncomfortable sensations in a lift are so insignificant that I could not draw a fixed conclusion therefrom. But upon a toboggan slide I determined with much greater certainty that riding down the steep descending track with head in upright position produced a very marked discomfort in the stomach, while this discomfort was totally lacking with the head inclined forward. A well known ski runner tells me that he always holds his head forward during a quick descent, since he otherwise becomes seasick. Upon the above mentioned sea voyage I determined the following with absolute certainty: Lying upon my back or abdomen, I did not feel the vertical motion of the ship at all unpleasant, but when I held my head upright I immediately experienced nausea and was obliged to vomit. I repeated the experiment five or six times, always with the same result. This demonstrates, therefore, that nausea during seasickness, at least upon vertical ship movements, depends upon the excitation of a sensitive organ in the head, and only the otolith organ comes here into consideration.

Naturally, it is quite different when the ship rolls. These turning motions cannot include a greater angle than 180 degrees, and one can imitate the ship movements if one turns a man to and fro upon a turning stool at about 180 degrees. In this way, by holding the head forward, we can imitate the side-to-side motions of the ship, and by including the head toward the right and left shoulder one can imitate the ship's movements fore and aft. One thing, to be sure, is not taken into consideration, that is, the action of centrifugal force, which in these ship movements plays a great rôle and must exert a strong effect upon the otolith organs. If one tests the influence upon the semicircular canals, then it is clear that with the same kind of turnings up to 180 degrees no afternystagmus can arise, as one may conclude from the theory of Breuer. Neither Ruttin nor Neumann could find nystagmus

during seasickness. That in sensitive persons turning toward right and left may call forth nausea, I have myself proven. There are persons who are insensitive to the strongest canal irritation, but in whom otolith excitations call forth nausea, and on the contrary such persons experience nothing unpleasant by up-and-down movements, but with the head in upright position experience nausea through turning movements. In seasickness, besides the excitation of the vestibular apparatus, other matters also play a rôle, particularly bad odor, anxiety, etc., things which can produce an influence upon the vomiting center, and therefore can combine with the excitations which go out from the vestibular apparatus.

SYMPTOMS IN UNILATERAL AND BILATERAL LABYRINTH DESTRUCTION AND THEIR SIGNIFICANCE.

The symptoms of labyrinth destruction in animals and in mankind have been so frequently described that I probably do not need to go into them thoroughly here. As long as there was no functional testing of the vestibular apparatus, it was never certain that the observed symptoms really depended upon a destruction and not upon an irritation of the labyrinth. The first investigator who gave an impetus to functional testing was Breuer. He observed the symptoms arising after cocainization of the semicircular canals in pigeons, which were described by Koenig as loss of function. In order to determine whether the canal apparatus really no longer functionated, Breuer touched the exposed canal with a bristle, and observed whether a reaction resulted from the direct irritation. We have here a rough method of testing the fistular symptom. Breuer then determined that irritations and loss of function are differentiated only by the direction of the produced nystagmus movements. Without entering into an exact description of the symptoms following unilateral labyrinth destruction, I should like to discuss several of the symptoms a little more fully.

If we destroy the right labyrinth, then there arises, as is well known, in man, and also in animals, very strong rotatory and horizontal nystagmus toward the sound side. Movements of the head increase the nystagmus the first two or three days. Recently I was able to show in one case that it is the

turning of the head to the diseased side which strengthens the nystagmus. It would be important for the sake of theory if the influence of head movements upon the nystagmus should be exactly studied. The caloric reaction and nystagmus by compression and aspiration are naturally suspended upon the diseased side. I have seen, however, in cases in which there was still some reaction by compression and aspiration of air in the external canal, exactly the same strong nystagmus to the sound side as in total destruction. Moreover, Ruttin has repeatedly observed very strong nystagmus to the sound side in serous labyrinthitis when labyrinthine function was still present. Certainly a marked diminution of function brings about strong nystagmus to the sound side. No systematic investigations concerning the reaction to the sound side exists for this stage. I have recently in one case determined a marked diminution of the excitability to cold water at the time of most violent symptoms. Systematic investigations upon animals are also needed here. Certainly, different cases behave differently. It would be of great theoretical interest to compare the changes in excitability of the sound side in connection with the disappearance of spontaneous disturbances. As is known, spontaneous nystagmus gradually diminishes in intensity from the moment of labyrinthine destruction. This disappearance may occur immediately after a labyrinthine operation, or the nystagmus may completely vanish in from two to three days, but in other cases the nystagmus may remain unchanged for a long time. I am under the impression that this is especially true where slight meningitis is present. In such cases diminution of excitability of the sound side is frequently lacking. I have only accidental findings in support of this and have carried out no systematic investigations. The appearance and disappearance of spontaneous nystagmus after labyrinthine destruction surely depend for the most part upon central conditions. Voss has furnished proof of this in one case in which in a unilateral labyrinth destruction a canal on the other side was wounded through operation and the labyrinth was apparently immediately put out of commission. Here there took place, like in an individual with two sound labyrinths, one of which becomes suddenly functionless, nystagmus to the opposite side, that is, to the side without a labyrinth. This case demonstrates that the periph-

eric production of a spontaneous nystagmus upon destruction of the labyrinth is quite subordinate to its central production.

In the first few days of a spontaneous nystagmus more or less strong reaction movements are usually present. These reaction movements are differentiated from cerebellar disturbances in that they correspond exactly to the nystagmus, are always present in the same manner in the right and left extremities, and depend in a typical way upon the head position. The reaction movements of the body and of the extremities have not been systematically worked out in animals, especially with respect to their variability in different positions of the head, but just here a systematic investigation on a series of animals would bring about important conclusions concerning the structure of the vestibular apparatus of the cerebellum, and would furnish a knowledge of the different functions of the vestibular apparatus in animals and man. The reaction movements arising after labyrinth destruction in man usually diminish very quickly, and are completely gone in a few days. In rare cases they are to be elicited distinctly after eight or more days have passed.

Recent cases of bilateral labyrinth destruction without participation of the meninges, I have not yet had an opportunity to see. Voss, as is known, described such a case in Basel, which he saw three months after the acute destruction, in which no disturbances of equilibrium whatever existed. I explained in Basel that where normally strong vestibular reactions take place as a result of cerebellar irritation, tone disturbances of certain duration are to be expected in bilateral destruction, but where the reaction movements were normally very trifling, striking symptoms could scarcely be observed, even in bilateral destruction. I am convinced that most disturbances of equilibrium in cases of transitory healed meningitis are to be explained through participation of the cerebellum. The great sensitiveness of the cerebellar cortex to injuries is a further basis for this assumption. I have indeed seen several cases, as already repeatedly mentioned, in which there existed with a functionating vestibular apparatus disturbances of equilibrium of the same type as in bilateral labyrinth destruction resulting from meningitis. Moreover, Ruttin has reported such cases.

In a work of James, which I have recently again perused, the same opinion is pronounced. Possibly early testing of the pointing reaction, determination of spontaneous pointing errors with lost irritability of the peripheral vestibular apparatus, and independence of the mispointing reaction with respect to existing spontaneous nystagmus, could bring about important proofs of the cerebellar origin of disturbances of equilibrium in these cases.

INTRACRANIAL SPONTANEOUS NYSTAGMUS WITH ATTACKS OF DIZZINESS.

The spontaneous nystagmus which arises in diseases of the end organ is always rotatory and horizontal, with a single exception. In labyrinth destruction there is often a stage in which pure horizontal nystagmus toward the diseased side exists for several days. The explanation as to why the peripheral nystagmus, or the nystagmus from the nerve, always has this character is, I believe, the following: If there is produced a peripheral attack of dizziness through some irritation as yet unknown to us, then the pathologic rotation spreads itself out over the entire intact center of Bechterew's nucleus and affects all of the ganglion cells at the same time. This causes a rotatory and horizontal nystagmus towards the diseased side. The rotatory nystagmus is the result of the irritation of the cells controlling the vertical canals. The horizontal nystagmus is the result of irritation of the cells controlling the horizontal semicircular canal. Such an attack of dizziness, as Jackson has already shown, possesses great similarity to an epileptic attack, only that it remains localized to the vestibular apparatus.

In a similar manner the nystagmus arising in unilateral labyrinth destruction toward the sound side is to be explained. But if there exists an intracranial disease, one part or another of Bechterew's nucleus can become injured, and through this all possible forms of nystagmus may arise. For example, pure horizontal, horizontal toward the one and rotatory toward the other side, diagonal toward the right or left and vertical toward above or below. Moreover, the attacks of dizziness of intracranial origin lasting for a longer time may manifest themselves through especial forms of nystagmus. Thus, I

saw one attack with rotatory nystagmus toward the one side and horizontal nystagmus toward the other side, in view straight ahead. Attacks with vertical nystagmus may appear, if one may draw definite conclusions, from the descriptions of the patient and from the vertical nystagmus produced experimentally in the turning chair. A nystagmus of high degree existing for a long time unchanged is always of intracranial origin. I believe that such a nystagmus is not due to constant irritation, but depends upon destruction of certain parts of a nucleus, which in turn produces uninhibited function of antagonistic cell groups. Moreover, there may be transitory paralyses which likewise improve with the receding disease, as observed in cerebellar abscesses. Frequently, however, there remains, even after healing up of the lesion, the previously existent central nystagmus, as I have seen in two operated and healed acusticus tumors, the slow growth of which usually reduces the transitory paralysis to a minimum when hydrocephalus is not combined with the tumor.

If the nystagmus is not to be differentiated from the peripheral type, then one may determine the correct diagnosis from the relation of the reaction of the vestibular apparatus and spontaneous nystagmus, as Neumann and I have shown. If labyrinth destruction or paralysis of the vestibular nerve, shown by absence of the caloric reaction, exists on the right side, and if the spontaneous nystagmus is directed toward the right side, then this nystagmus is of intracranial origin; for if the paralysis of the vestibular nerve were alone the cause of the spontaneous nystagmus, then it would have to be directed toward the sound side. Moreover, spontaneous nystagmus directed toward the sound side does not necessarily depend upon paralysis of the vestibular nerve. If the latter condition obtains, then spontaneous nystagmus gradually diminishes in intensity, as is well known, until, at the end of two or three weeks, it is no longer present to a pathologic degree. If the nystagmus is of intracranial origin then it does not diminish in intensity. If there exists no paralysis of the vestibular nerve, a strong nystagmus may be produced toward one or the other side by the peripheral labyrinth, but this nystagmus is then only transitory and changes in direction and strength. If it remains unchanged two or three days, then it is of intracranial origin. Change in the strength of

the nystagmus, on the contrary, does not demonstrate its peripheral origin.

The nystagmus produced by the brain cortex forms a special chapter. There are at least three places in the cerebral cortex from which one may produce eye movements and nystagmus. In the anterior central convolution there is a center for common movements of the eyes and head toward the opposite side. The center for voluntary eye movements is situated in the gyrus angularis, but eye movements may be produced also in the visual cortex. The nystagmus arising in the beginning of epileptic attacks is, for the most part, associated with the turning of the head and eyes in the direction of the quick movement of the nystagmus; that is to say, away from the diseased side, as I have repeatedly determined. This demonstrates the central convolutions as the site of origin of the nystagmus. In a case observed by me in which there was superficial hemorrhage on the brain cortex, I was able to observe in the course of an hour an entire series of such attacks. Once I produced in a patient with tumor of the central convolution, through vestibular irritation, a convulsion associated with turning of the eyes toward the sound side and strong nystagmus to the sound side. Rhese reports an accident case in which he observed the same thing.

The changes in excitability of the vestibular apparatus are interesting and important, especially those which arise in intracranial diseases. Ruttin and, later, Hautant have described the increase of excitability for experimentally produced nystagmus in diseases of the posterior fossa. I have seen this both when the vestibular nerve was intact on the right and left sides and also when there was unilateral paralysis. Neumann has observed increased excitability only toward the diseased side in unilateral paralysis of the vestibular nerve. The increased excitability in the vestibular system probably represents only one stage of the disease process. Later, there occurs diminished excitability, as I, and Neumann, too, have observed in cases of acusticus tumor. We shall speak later of the increased excitability for reaction movements. The increased excitability for nystagmus manifests itself by the fact that turning ten times calls forth a nystagmus lasting an extraordinarily long time. Thus, I observed in one case with a unilateral paralysis of the vestibular nerve, a duration of the

nystagmus to the diseased side after turning ten times of more than forty-five seconds, while ordinarily the maximum duration to the diseased side lasts twenty-five seconds. Moreover, the nystagmus to the sound side in this case was very strong and lasted more than forty-five seconds. The increased excitability shows itself further in that the caloric reaction is called forth very quickly and is of great intensity and duration. As I have observed, the strength of the nystagmus in these cases is out of proportion to the subjective disturbances of the patient. Very frequently a feeling of dizziness and turning sensation is completely lacking, even in a strong experimental nystagmus and if spontaneous attacks of dizziness still exist. The patient often says that such attacks appeared only in an early stage. Nausea, at any rate, is not usually present. With the increased excitability the production of nystagmus attacks is not seldom caused by quick head movements. I have seen cases in which every movement of the head called forth an attack of nystagmus which lasted, for the most part, only a half minute, but which, in one case, lasted for two and three minutes. In several cases I was able to show that the direction of the nystagmus during such an attack depended upon the produced head movement. If, for example, the head is inclined backwards, then there arises, as is known, during this movement vertical nystagmus upward, which is inhibited at the moment of stopping. In the above mentioned cases, however, the inhibition failed, so that at the moment of stopping a nystagmus attack was observed with vertical nystagmus directed upward. Quite similarly, forward inclination of the head produced an attack of vertical nystagmus downward. Inclination toward the left shoulder produced a rotatory nystagmus toward the left, and inclination toward the right shoulder produced a rotatory nystagmus toward the right. Increased excitability and nystagmus attacks are in no way indissolubly associated with each other. Thus, I observed one case of cerebellar abscess in which at the height of the disease increased excitability and nystagmus attacks were present. Healing gradually took place, the nystagmus attacks vanished first, and later the increased excitability, until finally, after complete healing, normal excitability was present. I have even seen cases with diminished excitability with nystagmus attacks upon movements of the head.

INVESTIGATIONS OF THE VESTIBULAR EYE MOVEMENTS IN THE UNCONSCIOUS AND IN NARCOSIS.

I was the first to study vestibular eye movements in unconscious and narcotized individuals. Let us first take the unconscious into consideration. I have found that in slight disturbance of consciousness nystagmus is still quite distinctly produceable, usually associated with a deviation of the eyes in the direction of the slow movement. In deep unconsciousness the nystagmus is absent, and there is only a deviation of the eyeballs in the direction of the slow movement. It seems that one can test the reaction of the vestibular apparatus even in deep unconsciousness, for example, in meningitis or brain abscess, and thereby, as I have shown, obtain an opinion whether in meningitis the inner ear is concerned or whether, as in two of my cases, a cerebellar abscess or a cerebral abscess is present. If the caloric reaction is present the labyrinth is intact and a cerebellar abscess is much less probable than a cerebral abscess. This I have determined in one case in which there existed a chronic middle ear disease with marked suppuration, in an individual who several months previously had shot a bullet into his head, and in whom the question was whether the unconsciousness was to be attributed to cerebellar abscess resulting from the ear condition or to a cerebral abscess resulting from the bullet wound. On account of the normal caloric test I diagnosed a probable cerebral abscess, which was found at operation. In another case I was called to an unconscious patient, in whom there existed a deviation of the eyes toward the left, together with a chronic middle ear suppuration on the right side. The coloric test showed lack of excitability of the right labyrinth. By irrigating the left ear with hot water there arose a deviation of the left eye into the right canthus, but the right eye moved only half way. I diagnosed labyrinth suppuration, cerebellar abscess and meningitis on the right side. From the deviation of the eyes toward the left, which corresponds to a spontaneous nystagmus toward the right, I diagnosed cerebellar abscess. From the abducens paralysis I diagnosed the meningitis. Operation and postmortem disclosed the correctness of my diagnosis.

Recently Rosenfeld has applied the vestibular test to determine the depth of disturbed consciousness, and thinks that he

can differentiate between hysterical and epileptic attacks by means of the caloric test. In hysterical persons nystagmus may be present; in epileptic persons only the slow deviation. I do not believe that a thorough difference can exist. If one assumes that the quick movement of the nystagmus arises in a supranuclear center, then it is clear that even in complete inactivity of the cerebral cortex the nystagmus reaction must be retained, but if the loss of function attacks deeper centers also, the quick movement of the nystagmus may fail. It is quite certain that in deep unconsciousness deeper centers lose their power, as is shown by loss of pupillary reaction. As complicated as these relations are, just so complicated are the symptoms during narcosis. In slight narcosis one can produce nystagmus in many individuals, but even then the quick movement of the nystagmus is very irregular or completely absent. I have seen repeatedly the quick movement arise just at the moment of the return of consciousness, but this does not show that the quick movement arises in the cerebral cortex. For the centrifugal operation originating in the cerebral cortex may be necessary for conscious vision in order that the paralyzed supranuclear center may again come into activity. If the narcosis is moderately deep, then the quick movement always fails, while the slow movement is still present, but the eyes are no longer strongly associated, for it is known that in narcosis in general there are dissociated eye movements. If one tests the reaction in narcosis, one must guard against a confusion of the same with resultant spontaneous slow eye movements. It has already happened to me that I could not certainly decide in one case whether a reaction was present or not, since the existing spontaneous turning of the eyes, now toward the right and now toward the left, concealed the reaction symptoms.

TESTING OF THE REACTION MOVEMENTS AND BEHAVIOR OF THE SAME IN PERIPHERAL DISEASE OF THE VESTIBULAR APPARATUS AND IN INTRACRANIAL DISEASES.

In testing the reaction movements, we must differentiate between those of the extremities and those of the body.

(1) Testing of the reaction movements of the extremities. The test of the reaction movements in the shoulder joint for deviation toward the right and left I have already described in the chapter entitled "Anatomy and Physiology"; also the lateral

movements of the arms from right to left and vice versa, for the testing of reactions in a vertical plane are already there discussed. Just as we test the wrist joint, we can test also the shoulder joint reactions in different positions of the arm and of rotation inwards and outwards, and we find not rarely a disturbance only for one position. We have further to test the movements in the elbow joint. This is done very simply by supporting the elbow in some manner and, with the wrist joint fixed, allowing the movements to take place only in the elbow joint again with pronated and supinated forearm. The test of the reaction movements in the wrist joint is thoroughly described in the anatomic-physiologic part. The reaction movements in the lower extremities one may likewise investigate separately in the hip joint, knee joint and ankle joint. I have, however, quite frequently found a loss of reaction movements of both extremities in normal individuals, and also in pathologic cases. The reaction movements of the same on both sides were very trifling or were completely lacking. On this account I have, therefore, hitherto carried out especial tests of the knee joint and ankle joint only in a few cases, and have limited myself as a rule to test of the hip joint. In testing the pointing movements we must always differentiate (1) spontaneous disturbances, and (2) disturbances of the reactions upon vestibular irritation. The spontaneous disturbances may be (a) transitory, or (b) permanent. Temporary disturbances I observed in many cases in which a disease of a cerebellar hemisphere was possible: for example, in acusticus tumors, in cases operated upon for labyrinth disease which complained of dizziness, or in cases having attacks of dizziness. These disturbances manifest themselves in the fact that an individual who usually points correctly with the finger, suddenly points past the finger. This pointing past continues several minutes and then disappears. We may assume that we here have to do with a convulsion of a given part of the cerebellum, that is, with a kind of cerebellar epilepsy. It is quite clear that if, for example, the center for movement of the arm toward the right suddenly transforms its potential energy into kinetic energy, then pointing past must take place towards the right. More exact investigations concerning the excitability during such an attack I have not yet been able to make. Permanent mispointing takes

place where a center for movement in a given direction is destroyed. If, for example, the center for movement toward the left is destroyed, then mispointing must take place toward the right. This is evident if we imagine the following simple conditions: In a state of rest the centers for right and left movement are in a normal indivdual held in a state of equal tonus, and if now a movement from above downward is carried out, then these two centers hold the balance so that they work upon the arm like two reins. The two are equally stretched. However, if one center is destroyed, one rein is lacking; then a deviation must take place in the direction of the other rein. If the destruction is permanent, then the pointing past should take place for life, as one may believe a priori. However, as a matter of fact, this is found only under especial conditions. If a center for movement in one direction is detroyed so that in no direction of the nystagmus and in no position of the extremities a reaction can be observed, then there always takes place through the cerebrum a compensation so that the spontaneous mispointing ceases after a short time. On the contrary, if the destruction concerns only one or another position of the extremity, and if a reaction is still retained in one or another nystagmus direction, then the spontaneous mispointing may persist for a lifetime also. Those cases are especially interesting in which only in one given position of the hand the reaction in one direction in the wrist joint fails. Hitherto I have seen about twelve such cases in whom the reaction in the wrist joint by position of the palm downwards, that is, by pronated forearm, was lacking. On the contrary, by upward position of the palm, that is, with supinated forearm, the reaction was present. In these cases the spontaneous pointing movement in the wrist joint gave a certain result, so that I could diagnosticate repeatedly according to the result of the spontaneous pointing movement, lack of the one and the presence of the other reaction. If, for example, in such a case, I tested the pointing movement of the right wrist with the palm turned downwards, then the patient pointed correctly. If now I turned the arm quickly over, so that the palm looked upwards, correct pointing usually resulted; I carried this out two or three times, then I pronated again and tested quickly the reaction, which now suddenly showed a strong spontaneous error, for example, toward the

right. In such a case, with the palm directed downwards, the reaction was lacking during the nystagmus toward the left. The explanation for this symptom lies, as I believe, in the fact that in the more accustomed position of the hand with pronated forearm the cerebral cortex compensates the error of cerebellar innervation, on which account no mistake arises upon carrying out this movement. If I then take the unusual hand position, palm turned upwards, one might expect that the cerebellar cortex would make an error too much toward the left. In fact, I could determine in single cases, even for the first movement with the palm upwards, a certain deviation toward the left. But if I now allowed the movement with the palm upwards to be often repeated, then the cerebrum made up for this error and the hand pointed correctly again. But if I do the opposite thing, then the cerebrum does not immediately compensate for the lacking cerebellar innervation, and on this account spontaneous error arises toward the right. In these cases a complete substitution of the cerebrum for the cerebellum cannot take place, because only in certain positions of the arm is the cerebellar innervation present. But if we have to do with a complete paralysis of the center in the cerebellum for the movement toward the left in which the reactions fail for all hand positions, the cerebrum may fully compensate, and thus spontaneous error completely vanishes and only the test of the reactions during nystagmus may here demonstrate the absence of the cerebellar innervation.

The spontaneous error which I have hitherto most frequently observed has always been directed in the right upper extremity toward the right and in the left upper extremity toward the left; that is to say, away from the middle line of the body. In a few cases, however, I have found also a spontaneous mispointing toward the middle line, and in others a fault upward. In most cases the pointing error was limited to the upper extremity, but several cases have erred in pointing with the foot of the same side toward the same direction as the arm.

The test of the reaction movements during nystagmus one may carry out by means of turning or irrigation. Sometimes one obtains stronger reaction by turning, sometimes by irrigating. An essential difference consists in the fact that in turning the reaction takes place in both labyrinths, but by the

caloric test only in one labyrinth. One may, therefore, conclude that in interrupting the connections, for example, from the right vestibular nucleus to the left cerebellar hemisphere which a median section of the cerebellum must produce, the reaction movement of the contralateral extremity after irrigation should be lacking. After turning, however, it must be retained. Such a case I have not yet seen. Moreover, cases with tumors in the gray matter of the vermis showed typical reaction movements of both extremities by caloric irritation.

In most cases observed by me the reaction movement failed in the upper extremity toward the middle line corresponding to the spontaneous pointing error away from the middle line. The possibilities of diagnosis are:

1. Acusticus tumors;
2. Cerebellar abscesses;
3. Traumas of the skull;
4. Cases of indeterminate etiology.

It is comprehensible that cerebellar abscesses and acusticus tumors produce the same result, since they affect nearly the same regions of the cerebellum.* But just why injuries to the skull produce these symptoms is not yet clear. This can be determined only postmortem. By temporary cooling of the cerebellar cortex after Trendelenburg, I could determine directly in man the center for the movement of the arms and leg toward the left. The person investigated had been operated upon by me for a right-sided cerebellar abscess, and was completely healed. The cerebellum behind the ear was laid free at the operation over an area about the size of a silver dollar, and was covered only by dura and thin skin. I carried out the investigation by temporary cooling very carefully, using water of 20° C., then of 12° C., and after I had proved this degree of cold as useless, but as completely uninjurious, I changed to ethyl chlorid. A half minute freezing of the skin behind the ear produced likewise no reaction; but after I increased the time from two to three minutes there arose a dis-

***Since the above was written, Docent Orzechowski of Lemberg placed at my disposal the history of a man with a right sided cyst of the cerebellar hemisphere, operated upon and examined postmortem, in which the right arm showed a spontaneous pointing error toward the right and the reaction by the caloric test failed toward the left.**

tinct action. The patient, who previously had pointed quite correctly with both arms and legs, suddenly pointed past toward the right with the right arm and leg, and when I now tested the movement toward the left of the right arm and leg by turning the patient ten times toward the left, it was seen that the right arm and right leg now did not deviate toward the left, which they had previously done promptly. The effect of the cooling lasts two to three minutes after one has interrupted the spray. The investigations, therefore, extended over a long period of time, for one can freeze a part only once at each sitting in order not to endanger the skin, and can only determine one or two facts. By repeated testing of the subject on different days, in the course of several weeks I succeeded in determining the following: (1) During cooling no trace of nystagmus exists; (2) the left arm and the left leg remained completely uninfluenced and showed after turning typical reactions toward the right and left; (3) immediately behind the ear lies the center of the movement of the right arm toward the left; for when I cooled off this region there arose a pointing error of the arm toward the right, and after turning toward the left, the reaction failed toward the left; (4) immediately behind the arm center is found the center for movements of the right foot toward the left. When I cooled this region the right arm remained uninfluenced, but the right foot now pointed past toward the right, and the reaction toward the left in the right foot after turning toward the left was lacking. Further investigations in this case, and as I hope in cases where the cerebellum is laid free over a greater area, will make clear the site of the centers for the remaining direction movements and joint movements. I believe that I may already say on the basis of pathologic findings that the center for the movement of the elbow and wrist joint toward the left must be in front of the arm center on the posterior surface of the pyramid.

I have seen with Sir Victor Horsley three cases after cerebellar operations in which the reaction away from the middle line was lacking and the reaction toward the middle line was present. In the histories which were amiably placed at my disposal nothing was said as to which part of the cerebellar hemisphere the operation affected, so in this direction the cases are not especially valuable.

In four cases, however, which were observed only clinically, I have found out an absence of the pointing reaction downwards. The changes in excitability which I could observe in cerebellar diseases, and of remote influences upon the cerebellum in its relation to reaction movements, are very interesting. While in normal persons the reaction movements are for the most part quite distinct in the shoulder joint, although usually no especially strong deviation is observed, the reaction in the wrist joint in normal persons is not seldom lacking. Moreover, in the lower extremities further reaction movements not rarely fail. In diseases of the cerebellum, however, a very marked increase of the strength of the reaction movements takes place almost always, and in all cases which I have hitherto investigated, and where a cerebellar disease came into question. I have also been able to determine reaction movements in the wrist joint. On the other hand, I have found in cerebellar diseases in several cases only very trifling or even absent reactions in the feet. The stronger the normal reaction movements are, the more distinct is the error of the reaction in a given direction, solely in the extremity of the diseased side. In the above mentioned cerebellar abscess I could follow the increase of reaction movements at the height of the disease and the gradual decline of the same during healing very distinctly. While during the time of wound treatment strong reactions in both wrist joints were to be elicited and the reaction toward the middle line failed only in the right wrist joint, several weeks after complete healing further reaction in the wrist joint, both on the right and left, had vanished, and only upon very strong caloric nystagmus did one see still traces of reaction in the wrist joint arise. The increased excitability occurs not merely in diseases of the cerebellum itself, but also in other kinds of diseases; when through mediation of a hydrocephalus, for example, the cerebellum is injured in its function. Moreover, loss of function may be produced by remote causes. But this concerns, so far as I have seen, not all reactions in all positions of the head and arms toward one direction. The remote influence can be recognized by the fact that at times no loss of function is to be determined at all. In cerebellar tumors I have, however, seen loss of function change with varying subjective feelings, and it is seen here, as everywhere else in neurology, that a single symptom signifies very

little, and only the entire clinical picture and consideration of the entire course of the disease, the determination of the constant symptoms and their differentiation from the temporary symptoms, helps to a correct diagnosis.

Concerning the brain paths which in the reaction movements of the extremities are to be considered, I have spoken already in the anatomic-physiologic part; but, to repeat briefly, these paths pass through the brachium conjunctivum to the opposite red nucleus and through the Monakow's rubrospinal bundle to the anterior horn cells of the concerned cerebellar hemisphere of the same side. It is comprehensible that loss of reaction movements may occur not merely in the cerebellar disease, but also in diseases along the course of this quite lengthy tract. Moreover, a loss of the voluntary innervation from the cerebral cortex must annihilate the reaction movements. But, in complete paralysis these reactions cannot be tested, and in paresis they must be retained in the paretic musculature, as I have repeatedly determined. It may be that interruption of the frontopontine or temporopontine tract here brings about loss of function, although we know nothing at all of this as yet. The case of frontal lobe tumor which Neumann observed was not sufficiently exactly investigated. A case which Ruttin and I have seen showed at one examination spontaneous pointing error (Ruttin), and at another examination normal pointing and normal reaction movements during nystagmus (Barany). Thus the characteristic of remote action was given.

Now as concerns the interruption of the tracts passing out from the cerebellum. I was able to show repeated loss of function in pontine diseases and in diseases in the region of the red nucleus. Between diseases of the cerebellum itself and these fiber diseases there exist, however, marked differences. While in cerebellar diseases the movement in one direction alone is almost always paralyzed and several centers only seldom are concerned, because the destruction must already comprise a large area, in fiber diseases a suppression of several or even all reactions through a relatively small disease area is quite possible. This was especially true in two cases of hemiathetosis in which an area in the rel nucleus was probably attacked and all reaction movements were lacking in the spastic joints, while the free joints and other extremities showed

typical and strong reaction movements. In such cases extraordinarily complicated disturbances may take place, the study of which demands hours and hours of repeated investigation. In general, however, one may say: Where only one reaction in a given direction and in a given joint is concerned, there a cerebellar disease is certainly present. Where, however, numerous reactions in varying directions are lacking, there exists a disease of the fibers. I should like especially to emphasize that the loss of one reaction may cause no disturbances in the movements of the extremity, and that the most exact neurologic investigation may disclose no disturbances. Where, however, several reactions are lacking, there is usually a tremor and also adiadokokinesis (Babinski) is demonstrable.

II.—Testing of the reaction movements of the body is carried out in an analogous manner to Rhomberg's investigations. Moreover, here we may differentiate (1) spontaneous disturbances and (2) disturbances of the reaction movements during nystagmus. If the patient sways during Rhomberg's test, then the following is to be observed: One has to test and to look for spontaneous nystagmus, whether there exists relations between the direction of the swaying and the direction of the nystagmus. If the stroke of the nystagmus is toward the left, and the patient falls by upright position of the head toward the right, if it can be demonstrated that turning of the head changes the direction of falling so that the patient, when the head is turned toward the left and anterior, falls forward, and when the head is turned toward the right, falls backward, it is then highly probable that a disease of the peripheral labyrinth or of the vestibular nerve exists before its entrance into the medulla oblongata. If, however, there exist no relation between the direction of falling and the direction of the nystagmus, and if the falling is not influenced through turning of the head, then it is either of hysterical or cerebellar origin. Neurosis is usually quite easily diagnosticated through the influence of suggestion. One places the patient with feet together and eyes open and he does not stagger. One now acts as if he is about to test the pupil reaction and asks the patient to repeatedly open and close the eyes. One sees then that by a longer closing of the eyes, if the patient's attention is diverted, no swaying arises. I have repeatedly succeeded

in demonstrating neurotic disturbances of equilibrium in this simple manner. Cerebellar disturbances of equilibrium manifest themselves frequently in that the tendency to fall takes place in single regions of the body, and in that the trunk and limbs do not work together in unison. (Asynergie cerebelleuse, Babinski.) Further, there repeatedly occurs in these patients tremor of the entire body. Testing in accident cases frequently presents difficulties because neurotic stigmata may be mixed with cerebellar stigmata.

Testing of disturbances of equilibrium during an experimentally produced nystagmus is carried out chiefly through irrigation. One produces a caloric nystagmus which must usually be stronger than is necessary for the caloric reaction. Then one asks the patient to stand and tests him for the Rhomberg symptom. When the right ear is irrigated with cold water the patient must fall toward the right. The influence of the position of the head upon the direction of falling must also be tested. It would be of great advantage if one could continue the irrigation during the entire duration of the test. An easily managed apparatus is yet to be constructed. In normal individuals the reaction movements always take place in a typical manner. Their strength is, to be sure, very different. There are people who are very strongly inclined to fall so that one can hold them upright only by summoning all one's strength; others, again, show only slight reactions which scarcely can be recognized by the unpracticed. There are, however, normal individuals who do not stagger in the slightest, even during the strongest nystagmus.

According to Nothnagel, Bolk, and others, the vermis of the cerebellum controls the musculature of the trunk. Analogous to the reaction movements of the extremities, I assume that the reaction movements of the body are also controlled by the cortex of the vermis, and that in the vermis cortex various centers for moving the body forward and backward to the left and to the right exists. Just as we differentiate movements of the extremities we may also differentiate spontaneous disturbances and disturbances of reaction through vestibular irritation. We have already spoken about the spontaneous disturbances. These may be transitory or permanent. Transitory disturbances of equilibrium, which continue hours or days, I have seen chiefly in persons in whom a remote action

took place in the cerebellum through hydrocephalus. For the most part, falling backwards takes place here. Permanent disturbances I observed in diseases of the vermis. Here falling always exists in the same direction. In two cases in which the disease had begun at a very early age, I observed a compensation evidently due to the cerebrum. One case was a tumor of the right vermis operated upon by Sir Horsley, which showed no distinct spontaneous disturbances of equilibrium. The second was a boy operated upon by Fedor Krause, in whom he extirpated an enormous tumor involving the vermis and both hemispheres. This boy could stand even during the time he was obliged to wear a dressing, and showed very trifling ataxia of the extremities. Here the cerebrum had evidently gradually assumed the functions of the cerebellum during the slow development of the tumor. It is known that cases with absent cerebellum do not necessarily show disturbances of balance. A test of the vestibular reactions here would be very interesting. Permanent staggering in a given direction, like permanent mispointing, I believe may be traced to loss of the center for the contrary movement. One must remember that probably these centers are paired, and that even in destruction of one center a reaction can still be produced from the other center through vestibular irritation.

Loss of reaction movements during vestibular irritation I have observed to be permanent only in diseases of the vermis. Temporary loss and disturbances of reaction one sees quite frequently in remote action upon the cerebellum. One must differentiate between loss and disturbances of the reaction. If I irrigate an individual and he manifests a strong nystagmus toward the left and falls backward just as he fell before syringing, then I speak of a loss of reaction. If, however, with the head in an upright position he falls toward the right and backward, while he previously had fallen only backward, then a reaction is certainly present. If in such a case I turn the head toward the left while the caloric nystagmus strikes toward the left, then, as is known, the normal patient must fall forward, but if now he falls also toward the right and backward, then I can diagnosticate a loss of the reaction in this position of the head. For diagnostic purposes this loss of reaction is only of value when it is constant. It is not rare to find the patient falling forward on one day and on the next to

find that this reaction is absent. Then one may diagnose remote influence. In three cases I was able to diagnose vermis tumor through exact testing of the falling movements. My diagnoses were corroborated partly upon postmortem, partly at operation.

In two other cases I have diagnosed remote action, which was verified postmortem. Even if falling in a given direction upon irrigation of one ear is absent, it may be elicited from the other ear. This depends upon the fact either that the fibers of the concerned center are interrupted, or that right and left centers are present for the movement of the body, and that the right ear is connected with one center and the left ear with the other center. I can also produce falling forward and backward by turning the individual with the head inclined toward the shoulder and producing thus a vertical nystagmus in an upward or downward direction. If I then turn the head straight, there frequently arises, even when the patient is sitting in the turning chair, typical falling of the body. But here, of course, both labyrinths and both centers are excited, and one cannot exclude even in normal reactions a loss of one center.

As for the tracts through which the reaction movements take place, it is very probable that the fibers from the roof nucleus pass out to the nuclei of the vestibular nerve, and from here the vestibulospinal tract descends to the anterior horn cells. It is, therefore, comprehensible that also diseases in the region of the vestibular nucleus could give rise to spontaneous falling and loss of body reactions. It is very remarkable that the root fibers of the vestibular nerves give so many collaterals to the nuclei of the vestibular nerve, and that the reaction of falling should go over, not directly, from the root fibers to the nucleus and the vestibulospinal tract, but pass indirectly by way of the vermis cortex and the descending tracts from the cortex to the nuclei of the vestibular nerve. (Faisceau en Crochet, van Gehuchten.)

I cannot give a true explanation for this condition, but it seems to me possible that the root fibers maintain a kind of collective tonus of the body musculature, and that the centrifugal innervations from the cerebellar cortex bring about the change of this tonus, and call forth the falling movements

through a given vestibular irritation and a given position of the head in a different direction.

Further exact anatomic and clinic observations may be of value here. We ought not to forget that we are only just beginning to investigate cerebellar function, and that it is impossible that our experience hitherto should lead us to absolutely certain conclusions.

III.

THE RELIEF OF PAIN IN ADVANCED TUBERCULOSIS OF THE LARYNX BY MEANS OF INJECTIONS OF ALCOHOL INTO THE INTERNAL LARYNGEAL NERVE.*

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The problem of the relief of pain is one that always has confronted the medical profession, and while only a symptom, it sometimes assumes the importance of a disease and has to be reckoned with and treated as such. This is certainly true in the great majority of cases of late tuberculous laryngitis, a patient suffering from this condition usually being a most pitiable object. The ceaseless ache or pain engenders restlessness, prevents adequate rest both day and night, and interferes most seriously with feeding, even to the point of causing semistarvation. Up to within the last few years there have been no satisfactory means of combating this suffering. Menthol, cocain, orthoform, anesthesin and other drugs have been used locally, but they have proven to be far from adequate, as their action is fugacious, they are difficult or impossible of application by the patient himself, and at times they all fail to produce any result whatsoever. The last resort is some form of opium, and while this should never be withheld if nothing else will give comfort, any other drug or remedial measure which will afford the same degree of relief, without the unpleasant secondary effects of opium, would be much more desirable.

HISTORY OF THE METHOD.

In a way the physician treating a case of advanced and painful laryngeal tuberculosis has to face the same situation that he does in cases of trifacial neuralgia, viz., pain is the

*Read before the John Morgan Society, Philadelphia, January 19, 1912.

dominant feature and must be relieved. About ten years ago Schlösser[1] introduced the method of interstitial injections of alcohol into the branches of the trifacial nerve, selecting usually the foramina of exit as the point of election for the injection. Since then the method has been used by many with noteworthy success. In addition to using this method in trifacial neuralgia, Schlösser has employed it in sciatica, and has never seen any serious accidents result as far as the motor fibers are concerned, while he has had good results as regards the sensory fibers.

One of the most recent articles on the subject is by Spear[2], who states that the results of such treatment are as follows:

"This form of treatment has been used in many hundred cases, both in this country and abroad. It has also been used over a period of nearly ten years. During this time we have been able to follow many cases so treated, and the result of the experience of these men, who have used this treatment in many cases, and over a long period of time, is that where the nerve is properly injected, in over ninety per cent of the cases immediate relief is obtained; that recurrences may occur in from eight to eighteen months; that some cases are permanently cured with one injection; that some cases require a second injection, and a few three or more injections; that with some experience this treatment can be readily carried out; that second injections are as efficacious as primary ones; that cases that have lasted many years are no more intractable to this treatment than more recent ones; that the patient's age is no contraindication to this treatment, patients ninety years old having been relieved by this means; that it should always be tried before more serious surgical procedures are instituted."

Another recent paper on the subject is by Kiliani[3], who states that he has used the Schlösser method in sixty-eight cases of neuralgia, and that all were rendered entirely free from pain for a certain time. In none had there been any hemorrhage, facial or oculomotor paralysis, or herpes. Two cases of ulcer of the lower lip resulted from the patient biting the anesthetic lip. In ten cases a drooping of the upper lip resulted from injections into the maxillary nerve, the drooping lasting from three to ten days, and in one case six months. In six per cent of the injections into the mandibular nerve, trismus of the masseter muscle prevented proper opening of

the mouth, this lasting usually one to three weeks, and in one case eleven months. Other unfortunate results have occasionally happened, Schlösser having reported cases of herpes and gangrene with deep sloughing.

The most recent contribution is by Patrick[4], who reports the results obtained in treating one hundred and fifty cases, and concludes that "For the very old, the very feeble, and for those with grave organic disease, I believe this to be incomparably the best treatment. Practically without danger, without shock, without an anesthetic, almost without inconvenience, the great majority of such patients may be kept comfortable until length of years or intercurrent disease brings the end."

No adequate explanation of the effect produced has been advanced up to the present time. In the discussion of an earlier paper of Kiliani's[5], W. B. Noyes asked "if there were much reaction or inflammation following the injection, and how Dr. Kiliani explained the relief that followed the procedure. Any radical method of affecting a peripheral nerve that was causing repeated attacks of intense pain, either of a spasmodic or a continuous nature, must either cause a degeneration of its nerve fibers, which was distinctly an interstitial change, or cause an accumulation of leucocytes in the neighborhood of the nerve trunk, either as an inflammatory reaction, as in counterirritation, and so reduce any perineuritis or neuritis proper." Kiliani had no explanation to offer. "It could be safely assumed, however, that the injection of alcohol produced a certain amount of paresis of the nerve, and during the course of the injection the patient described the gradual numbness following the distribution of the nerve. The numbness disappeared in the course of a few days or weeks, but there was no return of the pain."

In this connection Jeliffe[6] states: "A degenerative process is set up in the nerve trunk, which is recoverable, and general sensibility usually returns, but the pain is absent."

THE APPLICATION OF THIS METHOD TO THE RELIEF OF PAIN IN LARYNGEAL TUBERCULOSIS.

This originated with Hoffmann,[7] who suggested that the internal laryngeal nerve be injected with alcohol, and reported two cases in which he used the procedure. A second article on the subject was written by Levinstein[8], who two years later

reported six cases. The third, and the first to appear in this country, was by Lewy[9], and in it were reported two cases, thus bringing the number of reported cases up to ten. In addition to these there are reported herewith sixteen additional ones, five of them private cases, and the others from the White Haven Sanatorium and the Henry Phipps Institute of the University of Pennsylvania. In the latter cases some of the injections were given by Drs. I. W. Brown, E. E. Wisehart and W. A. Smythe, to whom I wish to express my indebtedness.

THE INTERNAL LARYNGEAL NERVE AND ITS SURGICAL ANATOMY.

This nerve lends itself peculiarly well to injection, in that it is purely sensory, it supplies all of the lower and the greater part of the upper larynx, it is comparatively near the surface and very accessible, and it is fairly well removed from important vulnerable structures. Branching off from the superior laryngeal it passes obliquely downward and inward to pierce the outer portion of the thyrohyoid membrane. Having passed through the membrane, it supplies all of the interior of the larynx with sensory fibers with the exception of the epiglottis, in whose supply the glossopharyngeal shares. Abolition of the function of this nerve will, therefore, check all pain sensations in the larynx except some of those originating in the epiglottis. As the latter is the part most accessible for removal, we have by a combination of injections and ablation ready means of dealing with pain originating in any part of the larynx.

In order to secure exact data in regard to the surgical relations of the nerve, dissections of formaldehyd-hardened bodies were made in the laboratory of anatomy of the University of Pennsylvania; for these I am indebted to Dr. P. G. Skillern, Jr. Hardened cadavers were used in order to secure conditions as they are found in vivo, and not as they are after the shifting, stretching and general mutilation which supervene on dissection of the ordinary soft body.

Overlying the nerve at the point of election for injection with the technic described below are skin, superficial fascia, platysma muscle and a dense layer of deep cervical fascia. Beneath these structures is a fossa like space which lies slightly below the center of the carotid triangle and is filled with loose

areolar tissue. This region is bounded as follows, the various structures in each boundary group being enumerated in order from the surface inward: Posteriorly, the sternocleidomastoid muscle, the facial vein and the superior thyroid artery, the carotid sheath lying just behind the latter; anteriorly, the posterior margins of the omohyoid and thyrohyoid muscles, the former slightly anterior to the latter; superiorly, the inferior border of the submaxillary gland and the hyoid bone; inferiorly, the upper border of the thyroid cartilage. Its floor is formed by the thyrohyoid membrane, over which run the internal laryngeal nerve and the superior laryngeal vessels.

The nerve in its course over the membrane describes a distinct downward loop, the lowest part of the curve reaching quite to the upper margin of the cartilage. In making the injection, therefore, the needle should be directed toward the lower part of the space, viz., along or near the upper border of the thyroid cartilage.

THEORETICALLY POSSIBLE INJURIES.

By inserting the needle too far back the following structures might be injured: The superior thyroid artery, the terminal part of the facial vein or the external carotid artery. If the insertion were too high the submaxillary salivary gland might be entered, or going still deeper, the hypoglossal nerve might be infiltrated. If a point too near the median line were selected, the injection would go into the substance of the omohyoid or thyrohyoid muscles, and through the medium of the deep fascia of their sheaths, the alcohol be prevented from properly infiltrating the nerve. If the injection were too low, the omohyoid and thyrohyoid muscles and the thyroid cartilage would receive the solution.

There is always present the fossalike region mentioned above, and into this the needle should always go. One can be reasonably sure of having entered this space by noting the great freedom with which the needle can move laterally in all directions. If it is inserted beyond the confines of this space this freedom of motion is greatly restricted.

THE SOLUTION USED.

This is a seventy-five per cent ethyl alcohol solution, to which cocain hydrochlorate is added to an amount sufficient to

make a one per cent solution. The cocain diminishes markedly the burning complained of when alcohol alone is used.

TECHNIC.

The technic used is practically that recommended by Hoffmann. The patient is placed in the recumbent position with a pillow under the shoulders and the head extended and rotated away from the side of the neck to be injected. (Levinstein administers his injections with the patient in a sitting posture.) With an anilin pencil the lateral part of the thyrohyoid interval is explored until a tender spot is found and marked, this spot corresponding to the internal laryngeal nerve. With the thumb and fingers of the left hand the larynx is steadied and then the needle is introduced at a right angle with the surface through the skin. After traversing the skin the needle seems to enter a cavity in which its point can move freely. It is then gradually pushed deeper and moved around in an attempt to elicit a pain in the ear, which is supposed to indicate that the point of the needle has come into contact with the nerve. In very few of my cases has this reflex been elicited, and when it does not appear, I simply push the point of the needle to the depth of a little less than 1 cm. The first push on the piston of the syringe is intended to deliver but a drop or two. This is done in order to determine by the patient's coughing whether or not the point of the needle has pierced the laryngeal mucosa and entered the lumen of the larynx. If all is quiet, a few more drops are forced out and the resulting burning sensation allowed to disappear. This takes but a minute or two, and then the remainder of the syringeful is slowly ejected into the neck, a total of twenty to twenty-five minims (1.3 to 1.6 cc.) being used for each injection.

EXPERIMENTS ON THE CADAVER.

In order to secure an ocular demonstration of the destination and distribution of the injected alcohol, I injected fifteen minims (1 cc.) of a saturated solution of methylene blue in alcohol into the necks of nine cadavers. After a lapse of several days the injected regions were dissected and the internal laryngeal nerve isolated. In seven of the nine the nerve was found to be stained a deep blue, in one a light blue,

and in one there was no stain at all. Further dissection of this last specimen revealed the presence of the injected solution in the interior of the larynx and pharynx, the needle having been forced all the way into the larynx.

The staining in the successful cases, of course, was not confined to the nerve, the surrounding muscular and fascial tissues sharing it. The area of infiltrated tissue varied from one to two inches (3 to 5 cm.) in diameter.

The results of these experiments were very gratifying, since without any aid in the way of an aural reflex or a tender spot, and simply from anatomic topography, eight of the nine were infiltrated, and in the ninth an accident occurred which could and would be avoided in the living patient.

REASONS FOR FAILURE.

In case the injection fails to bring relief, even after a second or a third attempt, there are several reasons which may be given in explanation. One is that the injection may not have been made at the right place, or that the needle had not been introduced to the proper depth. It is almost inconceivable that a syringeful of alcohol injected into the proper tissue plane within one-third of an inch (1 cm.) of the nerve would not infiltrate it sufficiently to cause it to cease functionating. My experiments on the cadaver would confirm this. If, however, the proper fascial plane be not reached, it is readily understandable that a dense layer of interposed fascia would prevent the alcohol from soaking through into the nerve. A second cause would be that the main seat of the pain lay in the epiglottis in the region supplied by the glossopharyngeal nerve. A third cause would be some anomaly in the course of the nerve, such as its entrance into the larynx through a foramen in the thyroid cartilage instead of through the thyrohyoid membrane.

UNTOWARD RESULTS.

These were noted in four cases. In one there supervened after an injection into one side only an insensibility of the larynx with retention of sputum. This patient was almost moribund at the time of injection. She died three days later, and it is not at all certain that the injection had anything to do with the sputum accumulations.

In a second case there occurred paralysis of half the tongue, on the injected side. This patient, some months previously, had a tuberculous ulcer at the tip of his tongue, for which a wedge at the end of the tongue was removed. While the paralysis caused no marked inconvenience, it was still present at the end of three weeks.

In two cases there was great difficulty on swallowing, which lasted three and five days respectively. In these cases both sides of the neck were injected at the same time, a distinctly unwise procedure. I would always allow at least three days' interval in case injection of the nerve of both sides is required.

TABLE OF CASES.

There have been so few cases in which this form of treatment has been employed, that a brief account of each may be of value.

No.	Reporter.	Age.	Sex.	Tubercle Bacilli in Sputum.	Laryngeal Lesions.	Pulmonary and General Condition.	Number of Injections.	Side of Injection.	Results.	Remarks.
1	Hoffmann	?	?		Edema of epiglottis and of right arytenoepiglottic fold. Ulceration and infiltration of both ventricular bands, both vocal cords and interarytenoid region.		1	R.	Swallowed food without pain. Could drink with difficulty, the best being tea with milk through a tube. Gained 1 lb. in 14 days.	
2	Hoffmann	?	?		Same as above.		1	R.	Same as above. Gained 1½ lbs. in 14 days.	
3	Levinstein	50	M.	+	Epiglottis and both arytenoids infiltrated and edematous. Miliary tubercles in epiglottis. Right cord reddened, thickened and superficially ulcerated. Ventricular bands ulcerated.	Advanced pulmonary tuberculosis. Pain so severe that taking of food was almost impossible.	1	R.	Spontaneous pain disappeared at once. Could swallow liquid and semi-solid food without pain. Only solid food caused pain.	

4	Levinstein	48	M.	+	Both arytenoids much swollen and the seat of miliary tubercles. Vocal cords and ventricular bands infiltrated and ulcerated. Arytenoepiglottic folds much thickened. Posterior laryngeal wall much thickened and the seat of an ulcer.	Emaciated. Advanced pulmonary disease. Severe pain on swallowing. Had eaten very little in two weeks, and then only with great suffering.	4	2 R. 2 L.	No improvement after first injection. Relief of pain after second; both of them on the left. Almost no pain on right side following first injection on that side. After second injection on right side could eat liquid, semi-solid and well cooked solid foods without notable pain.	
5	Levinstein	26	M.	?	Left ventricular band much thickened and ulcerated. Left cord reddened, thickened, superficially ulcerated and fixed half way between median and cadaveric positions. Posterior laryngeal wall thickened. Severe pain on left side.	Both apices involved.	1	L.	Permanent disappearance of pain in five minutes.	
6	Levinstein	31	F.	?	Epiglottis much thickened. Both arytenoids much infiltrated and the seat of miliary tubercles. On right arytenoid is a superficial ulcer with an irregular jagged margin. Ventricular bands and arytenoepiglottic folds much thickened.	Very feeble. Progressive pulmonary involvement.	3	1 L. 2 R.	Immediate relief followed the injection on the left side. No improvement followed the first injection on the right side. Pain disappeared from right side after the second injection.	

No.	Reporter.	Age.	Sex.	Tubercle Bacilli in Sputum.	Laryngeal Lesions.	Pulmonary and General Condition.	Number of Injections.	Side of Injections.	Results.	Remarks.
7	Levinstein	33	F.	+	Left arytenoid much infiltrated. Left ventricular band so infiltrated as to cover the cord of that side. Posterior wall of larynx thickened. Severe pain on left side on swallowing.	Progressive pulmonary disease. Marked pain on left side on swallowing.	3	L.	Pain was less after each injection and patient had entire freedom from pain after third injection.	
8	Levinstein	42	M.	+	Left arytenoid very much swollen and edematous; vocal cords congested and infiltrated; posterior laryngeal wall ulcerated.	Patient very weak with great pain on swallowing, especially on left side so that swallowing of solid food is impossible.	1	L.	Pain completely abolished even on taking solid food.	Before the alcohol injection seven submucous injections of novocain were given in the larynx but with slight effect.
9	Lewy	35	?	?	Infiltration of left arytenoid region, arytenoepiglottic region and left half of epiglottis, at the lower inner surface of which was a group of tubercles of which the visible area was about one-fourth inch in diameter.	Tuberculosis of lungs and spine of several years' standing. Pain in left side of throat for seven months. Severe for last week.	1	L.	Entire freedom from pain since the injection.	Ideal case.

10	Lewy	45	M.	?	Infiltration of epiglottis, arytenoepiglottic folds, ventricular bands and particularly of interarytenoid and arytenoid region. No ulceration.	Advanced pulmonary tuberculosis.	1	L.	Drank immediately 1 oz. of 20% alcohol. First comfortable swallow in two weeks. No return of pain in three days. Sensation of lump remains. Cough less and hence sleep is better. Feels touch of probe less on left than on right side of larynx.	
11	Fetterolf	30	F.	?	Epiglottis much infiltrated and on the right side is a deep ulcer. Both arytenoids and both aryteno-epiglottic folds greatly infiltrated, with an ulcer on the right arytenoid. Tuberculoma between arytenoids.	Advanced pulmonary disease. Great emaciation. Severe pain on swallowing for three months, especially on right side.	2	R.	Injection on right side was followed by considerable pain radiating toward lower teeth of same side. No improvement for thirty-six hours, then swallowing became easier. More difficult with liquid than with solid food. A second injection was given on right side two days later. Caused more pain than first. Following the second injection she swallowed with great ease.	
12	Fetterolf	32	F.	+	Huge infiltration of left arytenoid. Healthy stump of removed epiglottis. Slight infiltration of right arytenoid.	Advanced pulmonary disease. Very feeble patient. Intense pain on swallowing.	1	L.	Almost immediate relief Was able to drink within ten minutes with practically no pain. What pain there was was on the right side only. Next day she had her husband call me up to request an injection on the right side, as she had had so much relief from the injection on the left. Her throat remained comfortable till her death.	

No.	Reporter.	Age.	Sex.	Tubercle Bacilli in Sputum.	Laryngeal Lesions.	Pulmonary and General Condition.	Number of Injections.	Side of Injection.	Results.	Remarks.
13	Fetterolf		F.	+	Advanced tuberculosis of the arytenoepiglottic type, the epiglottis, the arytenoepiglottic folds, the arytenoid cartilages and the interarytenoid space all being very much thickened. No ulceration.	Extreme emaciation. Large cavities in both lungs. Intense pain on swallowing, in spite of taking about 4 grs. of morphine daily.	1	R.	Injection was followed at once by severe pain in the ear, which soon disappeared. No immediate relief to the pain on swallowing. Following day there was considerable relief but patient complained of stiffness and inability to swallow. On the next day the pain had disappeared, but the patient was so moribund that she had no power to expel the sputum which gathered in the trachea, larynx and pharynx. Swallowing was painless but the taking of food and drink caused violent attacks of coughing on account of their entering the larynx.	

14	Fetterolf		M.		Arytenoids swollen and congested, especially at their lower part; large erosion between the arytenoids, thickening and congestion of both cords, especially the right.		1	R.	Considerable pain during and following the injection, but ten minutes afterward could drink with absolutely no distress. Subsequently he began to get occasional twinges of pain, this apparently being due to extension of the laryngeal disease.	
15	Fetterolf	34	M.	+	Both arytenoids large as small plums. Epiglottis greatly thickened. Rest of larynx obscured.	Infiltration and cavitation of both apices. General condition fair.	3	2 L. 1 R.	First injection, left side, brought but slight relief. Second injection gave additional relief to left side. Third injection was on right side. For two days could not swallow. Now has no difficulty or pain in swallowing. Gained 3½ lbs. in five weeks.	
16	Fetterolf	32	M.	+	Infiltration of both arytenoids, with ulceration of the right.	Large cavity in left upper lobe. Infiltration of right upper lobe. Patient in bad condition.	2	1 R. 1 L.	Pain disappeared twenty minutes after injection on the right side and diet was taken freely for the first time in weeks. This lasted for two days, when the pain returned. Four days later injection was given into the left side with no results.	Patient died one week later.

No.	Reporter.	Age.	Sex.	Tubercle Bacilli in Sputum.	Laryngeal Lesions.	Pulmonary and General Condition.	Number of Injections.	Side of Injection.	Results.	Remarks.
17	Fetterolf	31	F.	+	Infiltration of epiglottis and of both arytenoids, with a large deep ulcer of the right arytenoid.	Infiltration and cavitation of both upper lobes. Patient profoundly emaciated.	2	1 R. 1 L.	Both injections given at the same time. Pain disappeared one hour after injections, but patient was unable to swallow without regurgitation through the nose. Two days later swallowing returned, but with difficulty, there being no pain, but much coughing.	Patient died sixteen days after injections, of acute dilatation of the heart.
18	Fetterolf	40	M.	+	Advanced tuberculosis of the larynx. Examination unsatisfactory as to details. Regurgitation of liquids for several weeks, and great pain on swallowing.	Consolidation of both upper lobes. Great emaciation.	2	1 R. 1 L.	Both injections given at same time. Pain disappeared one hour after injection. Partial paralysis of the throat followed and patient could not take more than one-half of the prescribed diet.	
19	Fetterolf	28	M.	+	Advanced tuberculosis of larynx. Examination unsatisfactory as to details.	Consolidation and cavitation of both upper lobes. Great emaciation and weakness.	1	L.	Pain disappeared one-half hour after injection. Patient took diet readily for a week, then pain on left side became very great. Patient begged for an injection on the right side, but on account of his dying condition it was not given.	Patient died twelve days after injection.

20	Fetterolf	25	F.	+	Extensive laryngeal disease, including ulceration of both arytenoids, and sloughing of epiglottis.	Extensive pulmonary disease and poor general condition.	8	5 R. 3 L.	Partial relief.	
21	Fetterolf	19	M.	+	Infiltration of epiglottis, left ventricular band, and both arytenoids with ulceration of last mentioned.	Infiltration and cavitation of lungs. Very poor general condition.	2	1 R. 1 L.	Complete relief.	
22	Fetterolf	44	M.	+	Involvement of entire larynx; ulceration of both arytenoids.	Infiltration and cavitation of both lungs; general condition poor.	1	L.	Marked relief for one day.	Patient died suddenly on day following injection.
23	Fetterolf	36	M.	+	Extensive involvement of larynx with ulceration of epiglottis and both arytenoids.	Extensive involvement and cavitation of lungs; general condition poor.	4	2 R. 2 L.	No relief.	
24	Fetterolf	26	F.	+	Ulceration of epiglottis, both arytenoids and both tonsils.	Both pulmonary apices infiltrated; general condition poor.	1	R.	Complete relief.	
25	Fetterolf	23	M.	+	Infiltration of both arytenoids, especially the right, and of the epiglottis.	Infiltration of both lungs.	2	R.	Complete relief after second injection.	Paralysis of right half of tongue followed second injection and remained for three weeks.

SUMMARY.

	Previously reported.	Writer's cases.	Totals.
Complete relief after one injection....	3	7	10
Complete relief after two or more injections	3	3	6
Partial relief	4	4	8
No relief	0	2	2

CONCLUSIONS.

Study of the reported cases would indicate that this method has a distinct place in the treatment of inoperable cases of advanced tuberculous involvement of the larynx. In it we have a procedure which requires no special apparatus or training, which is not hazardous or dangerous, which is not seriously painful, which can be repeated as often as may be required, which is usually successful at either the first or second attempt, and which can produce few untoward effects, and those but temporary. In the large majority of cases pain is relieved almost instantaneously, swallowing is rendered easier, a greater amount of rest and sleep is secured, nutrition is better maintained, the use of opiates is avoided and the natural cheerfulness and optimism of the patient are greatly enhanced.

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IV.

THE SUPERIOR MAXILLA—A DISCUSSION OF ITS PROPER DEVELOPMENT.*

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In preparing a paper on this subject at this time an apology is hardly necessary for reiteration of matter frequently before you in the current literature, because it is one of great importance, not only to those devoting their time to special work, but to the general practitioner, and, from certain points of view, to him rather more particularly.

It goes without saying, almost, that the configuration and development of these bones have features of import as great, if not greater, than any other of the bones of the body, because upon their proper evolution are dependent proper respiration, primarily, and, secondarily, nutrition, through digestion. It will not, then, if this be true, be much loss of time to recall to mind the anatomy of these bones and the functional purposes with which they are in relation.

Gray states that the superior maxillæ are the most important bones of the face on account of some of the diseases to which their parts are liable, and that "they are the largest bones of the face, except the inferior maxilla as a whole."

Each bone assists in the formation of three cavities: the roof of the mouth, the floor and outer wall of the nose, and the floor of the orbit; and also enters into the formation of two fossæ: the zygomatic and sphenomaxillary.

The body of the bone, hollowed in its interior, forming the antrum of Highmore, presents four surfaces, each of which contains certain structures of more or less interest to the purpose of this paper:

The external or facial surface shows: Eminences corre-

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sponding to the position of the teeth. Various fossæ, giving points of origin to facial muscles. Infraorbital foramen, the terminus of infraorbital canal.

The posterior or zygomatic surface presents: Several apertures leading to the posterior dental canals. A groove running obliquely downward and backward, forming, when articulated with the palate bone, a part of the posterior palatine canal.

The superior or orbital surface forms part of the floor of the orbit. Along the middle line of the orbital surface is a deep groove, the infraorbital, for the passage of the infraorbital vessels and nerve. This groove is sometimes covered so as to form a canal in its entire length. This canal subdivides into two branches, one of which terminates in the infraorbital foramen; the other, which is smaller, runs downward in the substance of the anterior wall of the antrum; it is the anterior dental canal and transmits the anterior dental vessels and nerves to the front teeth of the upper jaw. From the back part of the infraorbital canal a second small canal is sometimes given off which runs to the outer wall of the antrum and conveys the middle dental nerve to the bicuspids.

The internal surface. Behind the aperture leading into the maxillary antrum is a rough surface articulating with the perpendicular plate of the palate bone; it is traversed by a groove which, commencing near the middle of the posterior border, runs downward and forward, and when articulated with the palate bone forms the posterior palatine canal.

As the purpose of this paper is concerned chiefly in the effects of faulty development of the superior maxillæ, many points in the anatomic description have been omitted, attention being directed to the canals present therein. These are, to repeat:

Infraorbital vessels and nerve.

Infraorbital canal, terminating in the infraorbital foramen.

Canal for the middle superior dental nerve.

Canal for the anterior superior dental nerve.

Canal for the posterior dental nerves.

The posterior palatine canals, one large and several accessory.

The anterior palatine canals.

The nerves transmitted by these canals are:

The infraorbital canal transmits the superior maxillary division of the fifth nerve; in the canal, the middle and anterior superior dental branches are given off, which, as we have seen, traverse special canals in the substance of the bone to reach their points of distribution.

The middle superior dental branch is given off in the back part of the infraorbital canal and runs downward and forward in a special canal in the outer wall of the antrum to supply the bicuspid teeth. It communicates with the posterior and anterior dental branches, and at points of communication are thickenings, ganglionic in character.

The anterior superior dental nerve, of large size, is given off just before the exit of the nerve from the infraorbital foramen. It enters a special canal in the anterior wall of the antrum and divides into a series of branches, which supply the incisor and canine teeth; and a nasal branch which, passing through a minute canal, enters the nasal fossæ, supplying the mucous membrane of the forepart of the inferior meatus and floor of this cavity, communicating with the nasal branches from Meckel's ganglion.

The posterior superior dental branches arise from the trunk of the nerve just as it is about to enter the infraorbital canal, pass downward and enter the posterior dental canals on the zygomatic surface of the maxilla, communicating with the middle dental nerve and giving off branches to the lining membrane of the antrum and to each of the molar teeth.

The posterior palatine canals, for there is one large and several smaller, accessory canals, transmit the descending or palatine branches of distribution of the sphenopalatine ganglion. These branches are:

The anterior, or large palatine nerve, supplying the gums, mucous membrane and glands of the hard palate, the inferior turbinates and the middle and inferior meatus of both surfaces of the soft palate.

The middle palatine, supplying the uvula, tonsil and soft palate.

The posterior palatine, supplying the uvula, tonsil and soft palate, also, probably, the levator palati and azygos uvulæ muscles.

The nerves transmitted by these canals possess varying functions. Coming as they do from the Gasserian and Meck-

el's ganglions, they exercise sensory, vasomotor and trophic control, and, possibly through communication with the Vidian nerve, motor control.

Through one of the roots of the fifth nerve come the secretory nerves of the mucous glands of the entire nasal mucous membrane, as is proven by stimulating the trigeminus and producing secretion, while section diminishes it with consequent atrophic degeneration of the mucous membrane.

The vasomotor effects, so prominent in the symptomatology of diseases of the nasal mucosa, are derived through fibers coming to the Gasserian and Meckel's ganglia from the carotid plexus. The sensory function through the trigeminus proper and from the Vidian nerve, motor function to the levator palati and azygos uvulæ.

Let us now turn to the development of the maxilla and consider its ratio of development in relation to the other facial bones. Piersol states, concerning the superior maxilla, that "All the bones of the face except the lower jaw and the hyoid touch the superior maxilla. It has been described as the key to the architecture of the face. The palate bone both completes the palate and lies between this bone and the pterygoids, closing the posterior part of the opening into the antrum. The malar, joining the process of that name, makes the prominence of the cheek and helps to bound the orbit. The lacrimals and ethmoid touch the inner side of the orbital plate, and the ethmoid the inner surface of the nasal process. The frontal rests on the nasal process, and the inferior turbinate rests on the inner surface of the maxilla, and the vomer on the crest made by the union of the palate processes."

With such a relational situation it can be at once seen that the development of this bone is of the greatest importance. Its rapidity of growth in fetal life is far greater than the surrounding structures. It develops from four centers, which are deposited in membrane:

1. One which forms that part of the bone lying internal to the infraorbital canal, and includes the floor of the orbit, the outer wall of the nasal fossæ and the nasal process.

2. One which forms that portion lying external to the infraorbital canal and the malar process.

3. One which forms the palatal process posterior to Stenson's canal, and the adjoining nasal wall.

4. One which forms the front part of the alveolus and corresponds to the premaxilla of lower animals.

Of these, the third, forming the palatine process, is the largest and most important, producing as it does the major portion of the bone, and is directly concerned in the formation of the nasal chambers.

In a recent article by Hartz on the physiology and development of the nose, he states that the head and face grow more rapidly in two periods. The first is from birth to the eighth year. The second begins at puberty, when the head and face develop in all directions alike, and is completed about the age of twenty-one.

In the child the development occupies several stages. In intrauterine life the development of the head is far in excess of the face; during the first year the development of both head and face is nearly alike, though the face gains distinctively, especially in the lower portion of the nasal chambers. This is due to a downward and outward growth of the superior maxilla. From the second to the fifth year there is a distinctive gain in rapidity of the face over the head; the face becomes wider and the upper jaw more prominent. From the fifth to the seventh year there is further increase in facial development, the face becoming longer. With this development there are consequent alterations in the size and shape of the nasal cavities. At birth a line projected backward from the palatal process of the maxilla would intersect the junction of the sphenoid with the basilar process of the occipital. At three years this point of intersection would be about the middle of the basilar process, and at seven it would be about level with the edge of the foramen magnum. With this lengthening of the nasal chamber there is an accompanying lateral growth.

It is seen that these changes are a part of the early life of the child; that this stage of life is probably of greater importance than any other, because, unfortunately, there arise in this period causes which retard or arrest this development and thereby materially alter the shape, and, necessarily, the relational position, of the maxilla.

In making this latter statement it must be borne in mind that there are many who believe that deformities or retardation of development of the superior maxilla are due to the

peculiar type of skull. Siebenmann, Fraenkel, Haag and other German writers have expressed the opinion that the high arched and V-shaped type of alveolar arches depend on the type of the skull. The normal type is broad, with wide nasal cavities and dome shaped palate. The abnormal type has a high, narrow face, narrow nasal cavities and V-shaped alveolar arch.

It is not the purpose of this paper to discuss this question. Much can be said pro and con. Personally, I am of the opinion that nature never intended to promote a type which would so develop as to cause restriction of respiration, the most vital of all functions. I believe there is but one normal type, as above referred to, the wide nasal cavity, broad alveolar arch and dome shaped palate.

In studying a series of casts of the infantile pharynx, Braislin states that "In the newborn, shallowness of the nasopharynx is due to the undeveloped state of the bones of the cranium, face, and cervical vertebræ, chiefly the pterygoid processes of the sphenoid, the palate bones, the palate processes of the superior maxilla and of the vomer. * * * As age advances the growth of the bones of the cranium and face is very rapid, the vault of the nasopharynx becoming higher."

In considering the relationship of the maxillæ to surrounding structures it can be seen that certain forces might operate to retard the growth of the maxillæ while not affecting the bones with which they are united. I refer to the action of the facial muscles and of the tongue. We can see the possibility, therefore, of asymmetrical development, both as regards the maxillæ to their neighbors and to each other.

Cryer has made a great many dissections of skulls and studied the internal anatomy of the face, and his work has been of great value to rhinology. In reference to asymmetry he makes the following statement:

"There are even variations in the same individual in the shape, size and markings of the two sides of the face. In the bilateral bones, such as the frontal, sphenoid, vomer, ethmoid, and mandible, one side is usually found to differ from the other. In the homonymous bones, as the maxillæ, the malar, the lacrimal, the turbinate, and the palate bones, the same variations are observed. This being the case, it will be readily understood that the internal openings and spaces, as the

mouth, the nasal chambers, the orbits, the maxillary, frontal and sphenoidal sinuses, the ethmoidal and other cells, will differ accordingly."

With these facts in mind the importance of closely watching the growing child cannot be overestimated. Every factor tending to restrict development should be met and removed. Hypertrophied adenoids and tonsils are the chief factors for which our services are asked. Unfortunately it is the rule, at least in my experience, that the children brought to us for correction are, in the great majority of cases, from five years upwards. The children operated under this age are certainly fewer. It is still the impression among the laity, and with a great many general practitioners, that the child will outgrow these causes for his obstructed respiration. Possibly this is so, but in the meantime what is the result? These very imperfections of development which we all have seen and do see day after day.

It has been stated before that the most rapid period of growth is from birth to the fifth year. At this time development goes on with great rapidity, and it is during this time that any restriction of growth in one portion of the growing maxilla can produce effects which so alter the shape of the bone as to materially affect its relations and functions. Respiration, through narrowing of the nasal chambers, and mastication, through malocclusion of the teeth, are so interfered with as to cause serious impairment of the general health, and it is because of these effects that these cases are brought to us. There is another feature, however, not so generally recognized or considered, to which it is desired to bring attention.

In discussing the anatomy of the maxilla, particular mention was made of the canals, transmitting nerves, which passed through the substance of the bone or were formed by apposition with a contiguous bone. In normal development it is fair to presume that the lumen of these canals is free and unobstructed. In faulty development, however, variation in the course and size of the canal presumably occurs, with the possibility of stenosis and pressure upon the contents.

The hypersensitive nasal mucosa is almost the rule rather than the exception. We remove spurs, resect septa, reduce or remove turbinates and do various operative and treatment work on this region, in the great majority of cases, with ben-

efit; in some, however, with failure to relieve symptoms, and when failure does occur, is it not the experience of each of you that it is in the narrow type of nasal chamber that our usual procedures do not help?

We see many children in which widening of the face has not taken place because of failure of development of the palatal processes of the maxilla. As a rule, these children present the usual history of being weak physically, and frequently so mentally, and preeminently nervous and irritable. They are poor sleepers and show all the signs of irritable nervous systems. Repeated examinations may fail to show any particular cause. Adenoids may have been present and removed and the improvement that was expected has not taken place. Is it not logical to assume that this nervous irritability may be a reflex from a crowded nerve in a stenosed canal? Crowding of a nerve in a restricted or stenosed canal certainly should alter or impair its function, or by irritation lead to reflex phenomena. In an article on the effect of maxillary readjustment, Brown of Milwaukee says:

"Especially among growing children treated by this method has there been marked physical improvement, tendency to growth in height as well as general development and increase in weight. Many of these had been unable to attend school regularly because of tendency to nose, throat and bronchial affections. Nervousness was almost invariably greatly relieved, and this, it is believed, for two reasons: First, the well understood results from the improvement in breathing apparatus, with general healthfulness to be expected from better aeration and freedom from diseased nasal secretions, and, second, the relief from that condition to which Kiernan has called attention, caused by crowding together of the dental arches, with tendency to nerve irritation. This condition quite frequently manifests itself, not only in increased nervousness of a general character, but also in the development of neurotic tendencies leading to chorea, epilepsy and other similar affections, which, in some instances at least, might perhaps have been averted if these patients could have been tided over critical periods in their development."

Also, in a report of the Murray Hill School, a very interesting case is cited wherein the extraction of teeth, permitting readjustment and relieving irritation by crowding, produced

a remarkable change in the mental and physical condition of the subject. It is of such interest that I quote it in full.

Report of Miss Rowley, Murray Hill School, February 8, 1911:

In 1909 Carrie Mangino, 12 years old, a pupil of the Murray Hill school, was classed special four grade. She wore glasses to correct crosseyedness. Her manner in school was that of a nervous child. Her work and progress were hindered by some great strain. But there were glimpses of hope to tell the teacher that the girl had a good quality of mind, and that she could advance if some physical relief were given. October 18, 1910, Dr. Barnes examined her teeth. For six weeks thereafter her teeth were treated, and finally four teeth were extracted to relieve the impacted teeth. The week following December 17th, the time of extraction, a change in the girl's appearance is noticeable. Her eyes are straightened. In her own words, "I can now see things straight in front." Her nervous manner is gone. January 10th, she seems to be let down from some great strain or tension. January 17th, improvement in her lessons begins to show. She writes better. Her reasoning power is stronger. She has never read well enough to grasp the thought of a story; she does it now readily. Last year she was a poor, little, backward child to teach. This year the physical relief has made her a bright, animated, hopeful girl."

I also wish to cite a case of my own which has a like application to the point of view herein presented.

J. H., male, age five years, was brought to me for examination. He was a mouth breather, with all the attending symptoms, was excessively nervous, under size and poorly nourished. The lower border of the septum was partially deflected into the right nasal chamber, almost sufficiently so to obstruct respiration. His nasopharynx was filled with adenoids and the tonsils enlarged. There was marked arrest of development of the superior maxilla, as shown by the alveolar arch, the teeth being crowded and the bite very imperfect. I have a photograph showing this, kindly loaned me by Dr. C. A. Hawley, orthodontist, to whom he was referred.

The adenoids and tonsils were removed with some improvement in respiration, but not up to the normal standard. After a period of several months he was sent to Dr. C. A. Hawley, orthodontist, for readjustment. About two months later I

saw him again and the change was most marked; almost one-fourth of an inch space had been made between the central incisors. The respiration was wholly nasal, the nervous symptoms had disappeared and he had gained in weight and general appearance much improved. The septal deflection had disappeared.

In this instance the improvement in nasal respiration, so marked after adenectomy when there is more complete lateral growth of the maxilla, was not sufficient to give the relief which is usually looked for. The septal deflection, coupled with a narrow nasal chamber, still caused restricted nasal breathing. After a widening appliance had been used there was a very appreciable increase in the intranasal space with normal nasal respiration. But even with this the marked change in the nervousness was more than could be attributed to the respiratory improvement. With this there was, of course, improvement in the dental occlusion, which showed its effect by improvement in digestion. And yet, in spite of all this, I am of the belief that there was relief from reflex nervous irritability and that it was brought about by bringing to a more normal condition the paths through which the nerves of the nasal mucosa reach their region of distribution.

An hypothesis of this character would answer certain questions. We have seen at times, for instance, certain cases of atrophic rhinitis for which no adequate explanation as to cause could be given, except, possibly, heredity through some degenerate ancestor. It is more logical, however, to assume that if the trophic fibers which regulate the nourishment of the mucous membrane are inhibited in their function, we have thereby a cause rather than an indefinite empiricism which, by following its own law of practice, gives us no results.

This proposition also answers certain questions relative to cases of excessive irritability of the nasal mucous membrane that apparently resist those methods of treatment that in most cases materially benefit the patient; for it is also logical to assume that partial pressure upon a nerve may keep it in a state of chronic irritation.

When we look into the importance of the proper development of the maxillæ, however, when we realize that by reason of improper development so much harm may be done, it seems to me that the lesson of greatest importance to be learned is that of watchfulness of the growing child, to see that these

imperfections do not arise, and that if they should appear, to take immediate steps for their correction. It has been often pointed out that hypertrophy of the lymphatic ring around the nasopharynx is the chief factor that retards development; it must be especially impressed upon you that these causes should be removed in the early life of the child; the greatest mistake occurs in saying that these causes will disappear as the child grows older. It is true that they may, but it is also just as true that the damage has been done, and done to structures that are much more difficult to restore than the offending cause is to remove.

In the study of the maxillæ from this viewpoint, the following conclusions are reached:

1. That normal nasal respiration is only attained through proper lateral development of the maxillæ, i. e., the palatal processes.

2. That this development is materially retarded by those factors which prevent nasal respiration.

3. That in this abnormal development the shape of these bones may be materially altered, thus affecting their relations to contiguous structures.

4. That in this changed relation and altered shape we have factors for changing the direction and the lumen of the nerve canals traversing these bones, thereby making pressure upon their contents and causing alteration of function or, from irritability, reflex phenomena.

5. That no child is too young from which to remove causes for restricted nasal respiration.

6. That when abnormal development has occurred, readjustment of the maxillæ probably offers the best results that we can look for.

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V.

A VIRULENT AND RAPID INFECTION OF THE MIDDLE EAR AND MASTOID FOLLOWED BY AN EVANESCENT CELLULITIS OF THE NECK AND STREPTOCOCCEMIA.*

By John Randolph Page, M. D.,

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In view of the constant association of streptococcemia with thrombosis of the lateral sinus in the cases of mastoiditis reported by Dr. Gruening, based on the investigations of Dr. Libman and himself in 1908, and of the number of cases on the contrary reported by Dr. Duel and Dr. Wright, in which streptococcemia existed with mastoiditis without evidence of sinus thrombosis, this case will be of interest whether or not it be considered by some as the exception that proves the rule, and by others an example of the rule itself. At all events it is unique in being the only one of twelve cases of mastoiditis with bacteremia found by Dr. Strong in which thrombosis of the lateral sinus was not demonstrated later by operation. The infection occurred in a well nourished young man of twenty-two years of age, and the history of it rapidly given is as follows:

On the first of September, a few days after bathing in a crowded swimming pool, he awoke at six in the morning with pain in his left ear, which subsided to some extent during the day, but recurred the following evening and caused him a sleepless night.

The earache with neuralgia of the left side of the face and head, and slight dizziness, continued the following morning, and at eleven o'clock, about thirty hours after the onset, he came under my observation. At this time there was no discharge in the external auditory canal, and it was occluded in

*Read before the Section on Otology, New York Academy of Medicine, October 13, 1911.

its osseous portion by the dark blebs not infrequently seen in infections of this kind, so the drum membrane could not be seen.

Considerable tenderness was present over the mastoid antrum, but there was none over the rest of the bone, and his mouth temperature was 100.4°. Myringotomy was advised, and performed under nitrous oxid anesthesia at the hospital at one o'clock, and during this operation there was an audible escape of gas or air from the middle ear along with the fluid. A smear made from the discharge at this time showed a few streptococci as the only organisms present.

The discharge which followed the myringotomy was unusually profuse and watery, and of that brick-dust color sometimes associated with pneumococcus infections. A cultivation of it, however, taken from the canal was reported as staphylococcus.

The mastoid tenderness was not relieved by the myringotomy, despite the profuse discharge, and the temperature instead of subsiding rose to 103°.

On the following day the swollen condition of the canal and the patient's general appearance denoted a virulence of infection that indicated operation on the mastoid as the safest procedure, so the operation was performed that evening.

A dark unperforated cortex was found, and beneath it a pneumatic structure of bone, each cell of which was filled with dark material very like venous blood clot. A cultivation made from this material was reported as streptococcus. No exposure of the lateral sinus was made, and the inner plate was hard and solid. Except for the deep cellular structure in the region of the semicircular canals, which when it had been removed left them rather more exposed to view than is usually the case, there was nothing further of interest about the condition of the bone. The wound was washed thoroughly with salt solution, drained from the aditus to the lower angle with moistened gauze, and sutured to within three-quarters of an inch of this point.

It is worthy of note that on the day after the operation the patient had unusually free movement of his head and neck, felt more comfortable and his temperature ranged between 98.6° and 100.4°. That night, however, he complained of sore throat and pain on swallowing, but to all appearances his

throat was normal and his tonsils were not inflamed or tender.

At six o'clock next morning the temperature was 104.6°, and although there was no evidence of inflammation in the neck, or in the separated muscle at the tip of the mastoid, there was extremely acute tenderness, but no swelling or redness, beneath the anterior border of the sternomastoid muscle from the sternoclavicular articulation upward, the tenderness being as marked near the clavicle as above. There was apparently little tenderness except along the line of the internal jugular vein, and no redness or external sign of inflammation.

The wound was opened and washed out, but no evidence of infection was found in it, or in the neck in the region of it. Ice compresses were applied constantly along the jugular vein, and everything was in readiness for an exploration of the lateral sinus.

The next day the tenderness in the neck was less acute, and it had spread forward over the jaw and backward over the sternomastoid muscle to some extent, and for the first time a slight redness of the skin with a fine line of demarkation was discernible. The temperature did not go above 100.4°, and despite the positive report of streptococcemia which followed the blood culture made the day before, no exploration of the sinus was considered necessary. There was no further rise of temperature and the recovery of the patient was uneventful.

I learn from Dr. Strong, pathologist of the Manhattan Eye, Ear and Throat Hospital, that of sixty blood cultures made from cases of mastoiditis in that institution, twelve showed positive growths of streptococci, and of the twelve this is the only case in which thrombosis of the lateral sinus was not demonstrated by exploration of the sinus and resection of the internal jugular vein, and in which clinical evidence of sinus thrombosis was lacking.

The acute tenderness along the line of the vein, particularly in the lower part of the neck, was very unusual in my experience, and consequently alarming to me, but the appearance later of the redness and tenderness of the skin, though slight, away from the line of the vein, and the satisfactory temperature relieved my anxiety despite the report of existing streptococcemia.

The possibility that involvement of the sinus wall and vein occurred with spontaneous recovery is perhaps con-

ceivable, but it seems hardly probable with such a slight disturbance of temperature and general condition as there was in this case, the recovery of which was unusually strong and rapid, and I am interested to learn the opinion of the Section as to whether the very evanescent and apparently mild cellulitis is likely to have caused the streptococcemia.

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VI.

VACUUM NASAL HEADACHES WITH OCULAR SYMPTOMS ONLY.*

By Greenfield Sluder, M. D.,

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A great deal has been written in the past twenty-five years upon nasal headaches and their etiology. Some have been ascribed to contact; that is, when two mucous surfaces touch, as when septal deflections or ridges touch the turbinates (Harrison Allen, Jonathan Wright, and others); and many have been ascribed to suppurating accessory sinuses (Gruenwald, Hajek, and others). Some of these nasal conditions have been described with eye symptoms also; notably, the posterior ethmoidal and sphenoidal inflammations which attack the optic nerve by virtue of its nearness to these cavities (Birch-Hirchfeld, Posey, Packard, and Onodi); and influences have been thought to be exerted upon the interior of the globe by frontal and anterior ethmoidal sinus suppurations, in addition to the pain proceeding from such sinuses (Bull).

The class of cases described in this paper never has pus in the nose; never has the severe pain produced by suppurating sinuses; and never is complicated by blindness or changes within the globe. The eye disturbance is of the nature of "asthenopia." They are almost always closed frontal sinuses which are otherwise normal. Occasionally it is the anterior ethmoidal labyrinth that has become closed.

It is my desire in this paper to classify from a rhinologic standpoint, as far as possible, the origins of this class of cases, which was first described in two papers presented by Dr. A. E. Ewing and by myself before the American Ophthalmological Society, May 2, 1900.[1]

Ewing was the first to recognize these cases and to describe the symptoms, which briefly are: "Inability to use the eyes for

*Presented as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, January 1, 1911.

near work because of the headache which is produced thereby, and which is not relieved by glasses or eye treatment."

This class of cases is as a rule not accompanied by nose symptoms, unless they be produced by some lesion other than the one closing the sinus. Occasionally there is some obstruction to breathing.

CLINICAL PICTURE.

The headache is frontal usually; very rarely it is referred to the exterior angular process of the frontal bone. It is frequently present on rising, but grows worse on using the eyes; or is brought on by use of the eyes. The pain never reaches the intense degree of that produced by a confined empyema, but is quite sufficient to prevent the use of the eyes. Occasionally a patient relates that "blowing his nose is sometimes accompanied by a squeaking sound and sensation of air running up into his brain," which is followed very soon by temporary relief of the discomfort.

EWING'S SIGN.

The nasal trouble in these cases is revealed by tenderness of the upper inner angle of the orbit at the point of attachment of the pulley of the superior oblique and internal and posterior to it.[2] This is the portion of the orbit which is made by the frontal sinus, the wall of which at this site is thinnest. Kuhnt[3] observed that in empyema of the frontal sinus this was an exceedingly sensitive area, and suggested that this tenderness was in the supratrochlear nerves which were inflamed because of their juxtaposition. As a fact, it is in the bone at a point where the nerves are absent. It should be remembered that Ewing put this sign forth as a diagnostic help for cases which had up to that time been declared not frontal sinus cases, not nasal cases at all, because there were no nose symptoms, nor any pus, nor secretion from the sinus, nor any of the grosser commonplace anatomic changes. This sign is sometimes the only definite indication of the nasal trouble, the rhinologist's findings being negative.

The frontal sinus is most frequently the one involved because of the peculiar anatomy of its outlet. Rarely is the anterior labyrinth of the ethmoid the one involved, and then the symptoms are different and the tenderness is at the site

of the lacrimal bone. Patients affected this way have the feeling of sand in the eyes, and refer the pain to "behind the eyes."[4]

The posterior labyrinth of the ethmoid and the sphenoid, in my opinion, never give rise to these symptoms. They may give rise to occipital or parietal or frontal headache, or headache brought on by use of eyes because some of the ocular muscles arise in the apex of the orbit from parts made by the walls of these sinuses, or to blindness by reason of their nearness to the optic nerve, but never to this enduring low grade frontal pain with inability to use the eyes accompanied by Ewing's sign (see above).

Twelve years' observation of this class of cases leads me to believe Ewing's sign to be trustworthy and almost constant. The rare exception is in the case of the sinus having very thick walls, when the bones of the individual will in general be found to be very heavy and thick. Under these conditions I have found it absent even in acute empyema of the sinus accompanied by great pain.

GROSS ETIOLOGY.

In 1900[5] I stated that I believed the tenderness of the wall of the sinus arises secondarily to closure of its outlet, and that a similar condition obtains here to that produced in the middle ear by an acute closure of the eustachian tube. Many years ago this was recognized as a condition in which the oxygen of the enclosed air was absorbed and a negative pressure, a partial vacuum, established within the cavity. Brawley[6] concurs in this opinion. I believe this is the correct explanation, and that secondarily to the closure of the sinus, arises a congestion of the lining membrane in which the bone takes part to a degree which, however slight, is sufficient to render the thin wall of the sinus sensitive to external pressure —even to very slight pressure. The pulley of the superior oblique is attached to this thin wall. The function of this muscle being to turn the eye downwards and inwards, it is called into use for most of the acts of accommodation. So for close work there continues, more or less, a tugging at this tender point. This intensifies the dull ache made by the simple closure of the sinus. In cases of slight severity the patient is not especially uncomfortable until he begins using his eyes for close work.

The frontal sinus is the one by far most frequently the cause of this headache; according to my experience it being the origin of about 99 per cent of these cases. The attempt to classify the origins of these cases resolves itself, therefore, into a reference to the frontal sinus to that extent. In this paper I shall neglect the other sinuses. In 1900[7] I made no effort to do more than mention the ways in which the outlet might become closed. I did not report cases. I felt that my number of cases was too small. My material now comprises 451 case. These were nonsuppurative at the time they came under observation; and never at any time showed pus in the nose, nor did transillumination or X-ray examination show any clouding.

METHOD OF CLOSURE.

The frontal sinus may become closed in six ways, five of which I mentioned in my previous paper.[8] These subdivisions are recognizable by the ordinary examination. In the case of the normal nose the tenderness of the upper inner angle of the orbit (Ewing's sign) is assumed to be conclusive proof of closure of the frontal sinus because my experience has justified this conclusion.

1. The tubercle[9] of the septum, with or without a slight deflection of the septum, may be so large as to crowd the anterior third or half of the middle turbinate to the external wall, thus obliterating the space between it and the external wall, into which the frontal sinus opens by the hiatus semilunaris. This space from the anterior to the posterior tip of the middle turbinate, it seems to me, might be called the vault of the middle meatus. If I may be permitted to use this expression as a designation of this space, it will simplify these descriptions. Thirty-eight per cent of the cases were of this origin.

2. The vault of the middle meatus may be obliterated by the middle turbinate being simply lapped down against the external wall. Seven per cent of the cases were of this origin.

3. The vault of the middle meatus may be closed by hypertrophy of the middle turbinate uncomplicated by suppuration or polyps. Eleven per cent of the casese were of this origin. (Hyperplastic ethmoiditis, Uffenorde and Skillern.)

4. The vault of the middle meatus may be closed by swelling of its soft tissues (edema) without special hypertrophy of the soft tissues of the middle turbinate proper, that is, of the cavernous portion of the middle turbinate. Fifteen per cent of the cases were of this origin.

5. Normal noses. By this I mean that the middle turbinate is not hypertrophied; that the tubercle of the septum is normal; that the vault of the middle meatus is open. In this class of cases there is no history of empyema or of coryza as starting the trouble. These were the most mysterious until they were operated upon; that is, the middle turbinate removed. Then it was found that the hiatus semilunaris was narrowed or occluded, by bony narrowing, the uncinate process and bulla being in contact. It was this class of cases that I emphasized in the title of my paper in 1900. At that time I thought them to be more frequent than subsequent experience has proven them. The tubercle class I find outnumbers them. Twenty-four per cent of the cases were of this origin.

6. Empyemas or coryzas without suppuration which have gotten well, but have left a degree of swelling in the vault of the middle meatus sufficient to keep the frontal sinus closed and so keep up enough pain to render the eyes unfit for ordidinary work. Three per cent of the cases were of this origin. (These cases might be said to belong to the No. 4 set. Because of the etiology I prefer to separate them.)

In addition to these chronic cases one occasionally sees others that are excited by an acute coryza (without sinus suppuration) by reason of the swelling of the membrane in general, and subside with the coryza, spontaneously. I have construed these cases as having a small outlet from the frontal sinus, with the vault of the middle meatus normal. Swelling of the membrane in the vault from the coryza is sufficient to close the outlet, but as soon as the coryza subsides the swelling also subsides and the sinus opens; and then the symptoms subside.

DIFFERENTIAL DIAGNOSIS.

The supraobital nerve emerges from the orbit at the junction of the inner and the middle thirds of the supraorbital ridge. At this point it passes through the supraorbital notch,

and is distinctly accessible to pressure. Under normal conditions the site of this nerve is the most sensitive part of this area. The pressure tolerated by it normally is little. (In neuralgia of the supraorbital it becomes very much less.) The pressure tolerated by that portion of the orbit which lies internal and posterior to the supraorbital notch, which is made by the thinnest wall of the frontal sinus, is normally much greater than the nerve will tolerate. When the nerve is normal and the frontal sinus closed, the orbit interiorly and posteriorly to the supraorbital notch becomes as sensitive or more sensitive than the nerve itself. The supratrochlear as well as the supraorbital nerves are external to Ewing's tender spot. The supraorbital is more accessible for tests.[10]

HEADACHES FROM ALL CAUSES AND THEIR SEPARATION FROM THIS PRODUCED BY CLOSURE OF THE FRONTAL SINUS WITHOUT SUPPURATION.

Closure of the frontal sinus gives Ewing's sign (see above). This is not found in headaches produced by ocular, digestive, gynecologic, renal, gouty, neurotic, or any other cause except empyema of the frontal sinus.

PATHOLOGY.

The syndrome above described requires as a rule narrow noses for its development.

Class 1. (38 per cent.) The tubercle of the septum sometimes is so large as to crowd the middle turbinate over to the external wall of the nose on each side, producing the above described condition on both sides. As a rule, however, the tubercle is not so large—frequently normal in size—but is deflected to one side. The deflection of the septum is otherwise insignificant. With the aid of the tubercle it is very easy for it to crowd the middle turbinate to the external wall and close the vault of the middle meatus on that side. The histology of these turbinates is normal. The etiology of the deflected septum then becomes a factor in this class (Mosher,[11] Lack.[12])

Class 2. (7 per cent.) Middle turbinate lapped down tight to the external wall of the nose. I know of nothing special to suggest in reference to this class; because I know of no

reason why an otherwise normal turbinate should occasionally be turned so as to lap tight to the external wall without having been pushed into that position by the tubercle of the septum.

Class 3. (11 per cent.) Hypertrophy of the middle turbinate. The causes of hypertrophy of the cavernous tissues of the turbinate are discussed fully in the standard treatises on rhinology, to which the reader is referred.

Class 4. (15 per cent.) Vault of the middle meatus filled with edema of the soft tissues. This class bears a close resemblance to the polyp forming noses. Under the influence of an acute coryza these patients sometimes develop polyps. In the interval between coryzas the polyps disappear. The general edema of the vault of the meatus, however, does not subside. This condition has always seemed to me to be of the same pathologic origin as polyps, but of a lower grade.

In these patients in whom the middle turbinate has been removed I have many times seen, during a coryza, the hiatus semilunaris fill out with an edema and develop broad-base polyps in the hiatus and over the bulla, accompanied (without pus) by all the symptoms and signs of the original case. These would subside in a little longer time than is required for a coryza in a normal nose, and would not reappear until the next coryza.

Class 5. (24 per cent.) Normal noses. (To be considered below under Microscopic Pathology.)

Class 6. (3 per cent.) Empyemas or coryzas which have gotten well, but left a degree of swelling, sufficient to close the outlet of the sinus.

Clinically I have always felt that these cases bear a very close relationship to Class 4, that is, where the vault of the middle meatus is closed by soft swelling—low grade edema of the soft tissues.

MICROSCOPIC PATHOLOGY.

The middle turbinates which were removed in these cases are the material upon which the following observations were made by Dr. Jonathan Wright during the summer of 1909:

The turbinates of Class 1 and Class 2 showed little or no change.

The turbinates of Class 3 were not submitted to Dr. Wright. Nor were turbinates from Class 6, for the reason that I had no specimens. Dr. Wright's observations, therefore, bear chiefly upon the etiology of Class 4 and Class 5. Eight turbinates removed from cases of Class 4 showed some connective tissue in the stroma of the mucosa without any great activity in the bone changes, nor was the amount of bone increased. Some new bone formation showed at some places. Ten turbinates removed from cases of Class 5 showed thickened periosteum with great bone activity, active osteoblasts and few osteoclasts. The bone areas were large. Sometimes slight fibrous hyperplasia of mucous membrane was present.

TREATMENT.

It suggests itself, in logical sequence, that anything that may diminish swelling of the soft tissues, which close the outlet to the sinus, will be of service, and to this end I have employed the various astringents in commonplace usage, with satisfactory results for a very great number of cases, the applications being made in and about the middle meatus. My method of procedure is to try these applications in all cases. After a trial of two weeks, if there has been no response to treatment, more radical measures may be resorted to.

It is sometimes astonishing to see what may be accomplished by the simple application of astringents. In this connection I should like to cite the history of a lady, the wife of one of my intimate friends. She had suffered for five years from headaches and eye disturbances. She came under my charge in 1902. Her case belonged in Class 4. Following out the routine, I made an application of 2 per cent silver nitrate to those parts, about twelve times, extending over a period of three weeks, when the headache stopped, and has not returned. I state this positively, because my association with the patient socially gives me full opportunity to know.

It is well known that the obstruction to the outflow of pus from the frontal sinus is usually the middle turbinate; and so it is likewise the middle turbinate that is usually the obstruction to the ingress of air to the frontal sinus.

The more radical treatment is then the opening of the outlet of the frontal sinus, which is usually accomplished by the

removal of the middle turbinate. My method is to free the outlet to the anterior labyrinth of the ethmoid at the same time, removing the anterio two-thirds or three-fourths of the middle turbinate. It is my special effort to put my incision as high upon the capsule of the ethmoid (the external wall) as possible, about 2 mm. from the cribriform plate. In my experience this has been accomplished most readily by the method I described in the *Journal of the American Medical Association,* June 29, 1907. By this method the turbinate may be cut out up into the infundibulum. This seems to me also to be necessary, as it is at this place that the obstruction very frequently exists. In the very great majority of cases this suffices to open the sinus and thereby effect a cure; the headache stops; and the eyes go into unlimited service.

Three times I have found the uncinate process and the bulla of the ethmoid in a firm contact, reaching so high up that I was obliged to remove the uncinate process up to the level of the cribriform plate, and once some little above it.

Fifteen times I found the hiatus semilunaris filled out by a fibrous hypertrophy of the membrane covering the bulla; in these cases I succeeded in getting the result by a superficial galvanocautery destruction, the line of which ran parallel to, and just posterior to the uncinate process. The contraction of the scar resulting from this wound tends to pull the tissues out of the hiatus semilunaris. These were probably cases of hyperplastic ethmoiditis such as have been described by Uffenorde and Skillern and others.

PROGNOSIS.

The prognosis for the individual case is difficult. As shown by the case cited above, the simplest treatment is sometimes rewarded by a strikingly satisfactory result. On the other hand, cases that would apparently furnish a satisfactory result are sometimes found to be most stubborn. For these cases everything fails save the removal of the middle turbinate. This alone frees the inlet to the frontal sinus.

The prognosis for the operated case will be found to vary according to the microscopic findings in the turbinate which has been removed. If it shows marked periosteal thickening and bone activity, it will be found that in two, three or five

years the inlet will have so far narrowed, by the encroachment of the bulla upon the uncinate process of the ethmoid, that the patient will again have pain and a tender Ewing's point during and for a time following a coryza; and it may later come to pass that Ewing's point remains tender until the inlet has again been enlarged.

On the other hand, cases that show no bone or periosteal activity remain apparently permanently cured.

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VII.

DEEP TEMPORAL ABSCESS.*

By Alfred Braun, M. D.,

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If they are not evacuated artificially, accumulations of pus in the mastoid cells and antrum will eventually find an exit in one of several directions. The location of the exit may be divided into four groups: 1. Through the middle ear. 2. Through the walls of the external auditory canal. 3. Through the inner table. 4. Through the cortex. The location of the point of exit depends largely upon the anatomic conformation of the mastoid process, the size and location of the mastoid cells, the thickness and density of the cortex, etc.

In the first group, i. e., where the pus finds an exit through the middle ear, we may include:

(a) Those cases where the mastoid antrum and cells drain through a perforation in the membrana tympani. The pus in the mastoid can only drain through such a perforation when the aditus is unobstructed. In a large proportion of cases of mastoiditis the passageway of the aditus is blocked up by granulations. With a wide patent aditus and no adhesions in the tympanic cavity, the pus may very well drain through a perforation in the membrana tympani, and the mastoiditis get well without requiring an operation on the mastoid.

(b) The cases where the pus finds its way through the middle ear under the periosteum of the canal and on to the surface of the mastoid, simulating those cases in which there is a perforation through the cortex. The edges of the membrana tympani are very firmly attached to the annulus; yet it sometimes happens that pus from the middle ear may cause a separation of the membrana tympani from the annulus at one point, allowing the pus to burrow under the periosteum of the canal, finally forming a collection of pus under the periosteum of the outer surface of the mastoid.

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(c) The cases that perforate through the floor or anterior wall of the middle ear, following the areolar tissue about the eustachian tube, and forming an abscess in the soft palate, pillars of the fauces, or retropharyngeal space, the socalled "Senkungsabscess" of the Germans. These cases usually occur in chronic suppurative disease of the middle ear with extensive necrosis.

The second group of cases are the cases of chronic mastoiditis with necrosis and formation of a fistula in the posterior canal wall, or roof of the canal, through which the pus discharges. In some of these cases a large portion of the outer attic will and posterior canal wall is destroyed, so that almost a complete radical operation has been done by nature.

The third group, i. e., where the pus finds an exit into the cranial cavity through the inner table, may be divided into two groups:

(a) When the pus enters the middle fossa, and (b) when the pus enters the posterior fossa.

The perforation is apt to occur through the inner table in those cases where the cortex is thick and dense and the cells small and few.

The roof of the middle ear, aditus and antrum is composed of a very thin compact layer of bone. A perforation through this plate leads to the formation of a collection of pus in the middle fossa, resulting either in an epidural or subdural abscess, abscess in the temporosphenoidal lobe, or meningitis. Perforation through the plate of bone which lies over the lateral sinus and cerebellum leads to the formation of a collection of pus in the posterior fossa, resulting in perisinus abscess, sinus thrombosis, cerebellar abscess, or meningitis.

The pus may reach the posterior fossa by yet another route, i. e., through the labyrinth and internal auditory meatus. When the pus follows this path it results in meningitis or cerebellar abscess.

In the fourth group of cases, i. e., where the pus finds its way through a perforation in the cortex, there are three classes, depending upon the portion of the cortex perforated. These three locations are separated from each other by the attachment of two fasciæ to the bone, namely, the superficial layer of the deep cervical fascia overlying the sternomastoid muscle, which is attached to the outer side of the mastoid

tip, and the temporal fascia, which covers the temporal muscle and is attached to the linea temporalis and the upper border of the zygoma and malar bone.

The most common site of the perforation is between the attachments of these two fasciæ, on the outer surface of the mastoid process. When the cortex is very thin, especially in children, the perforation is apt to be located there. Sometimes the pus finds its way through an imperfectly closed squamomastoid suture. The pus lifts the periosteum and overlying fascia and skin before it, forming the well known subperiosteal abscess, which pushes out the auricle from the side of the head in a characteristic manner.

The next most common site is internal to the attachment of the superficial layer of the deep cervical fascia, i. e., through the inner surface of the tip, in the digastric groove. This occurs when the tip cells are very markedly developed. The pus here cannot reach the surface without perforating the firm, resistant deep cervical fascia. The pus is guided down beneath the fascia into the neck, where it forms an abscess under the sternomastoid muscle, giving rise to a deep swelling and tenderness at the side of the neck.

The least common location is above the lower attachment of the temporal fascia. A collection of pus in this location is known as a deep temporal abscess. In these cases the perforation is through the inner surface or root of the zygoma, or through the outer surface of the squama of the temporal bone. The entire zygomatic process is cellular in these cases, sometimes being converted into a hollow tube of bone. Sometimes the squama is separated into an inner and outer plate by cells extending upward from the antrum. In a colored girl operated on by the writer there was a cell extending upward for an inch and a half between the outer and inner layers of the squama.

The collection of pus in these cases lies under the temporal muscle, and is limited in extent by the attachment of the temporal muscle and its overlying fascia to the temporal bone.

The perforation is through the inner surface of the zygomatic arch. The manner in which the collection of pus reaches the location in which we find it at operation is shown in Figure 4. It does not perforate through the temporal muscle, but works its way back under the periosteum of the

zygoma to its root, and then forward under the periosteum covering the squama, or perforating through the root of the zygoma the pus pushes itself in all directions beneath the temporal muscle.

If these cases were left unrelieved the pus would probably work down through the sphenomaxillary fossa into the pharynx, as this is the only direction in which the abscess is not walled off by a fascial attachment.

Deep temporal abscess may come from other causes, but it is most commonly due to mastoiditis. The tenderness and swelling are located in front of and above the auricle. The tenderness is most marked over the arch of the zygoma. There may or may not be tenderness over the mastoid. The thing which is characteristic of these cases in the shape, size, and location of the swelling, which corresponds exactly to the shape, size, and location of the temporal muscle, this correspondence being accentuated by a marked edema of the temporal muscle itself, which always occurs in these cases. There is sagging of the anterior canal wall. The condition may easily be confused with a furuncle in the anterior canal wall, from which it should be differentiated by the following points:

In a furuncle the infection is under the skin and not under the temporal muscle, consequently the swelling does not correspond to the shape of the temporal muscle. There is not usually an involvement of the middle ear or mastoid tenderness with furuncle, although a mastoiditis may coexist with a furuncle. Movement of the cartilaginous canal does not cause pain in deep temporal abscess, but is decidedly painful in furuncle of the canal. The swelling in the canal is not tender to pressure in deep temporal abscess, but is in furuncle. In deep temporal abscess the swelling is usually limited to the bony portion of the canal, whereas in the furuncle the swelling is most apt to be located in the cartilaginous portion of the canal.

In order to evacuate the abscess it is necessary to continue the mastoid incision forward to the hairline, as shown in Figure 6, dividing the fibers of the temporal muscle and the underlying periosteum. The periosteum is best divided after elevating it from above the root of the zygoma, forward, over the squama. This maneuver will always bring to light the collection of pus.

The zygoma is often reduced to a hollow tube filled with pus, with a perforation through the inner wall. Sometimes, as in case 1, there is a septum of intact bone between the cells in the root of the zygoma and the cells in the zygomatic arch itself, so that one is tempted to stop the operation before all the diseased cells are eradicated. In removing the diseased zygoma it is well to leave some bony tissue, so as not to cause a sinking in of the cheek.

After the abscess has been evacuated the divided fibers of the temporal muscle and overlying skin may be united by sutures, and the cavity drained by a wick of gauze leading into the mastoid cavity.

Of the three following cases the first was operated on by the writer, in Dr. Berens' service, at the Manhattan Eye, Ear and Throat Hospital, and the second and third by Dr. Berens, who was kind enough to allow the writer to use the histories for this paper.

CASE 1.—Michael McL., a driver for the street cleaning department, 46 years of age, was admitted to the hospital on November 17, 1910, with a history of having had buzzing in his left ear for the past six weeks. Two weeks ago he began to have severe sharp pain in the left temporal region, and sometimes in the left ear. At times the pain stopped altogether. A week ago he noticed a swelling in the left temporal region.

His temperature on admission was 101° F. The left membrana tympani was red and bulging. There was a swelling of the anterior canal wall, especially marked in the deeper portion of the canal. This swelling was not sensitive to the probe. There was no sensitiveness on moving the auricle. No tenderness or edema over the mastoid. There was an edematous swelling above and in front of the left ear, corresponding in size, shape and location to the size, shape and location of the temporal muscle. This swelling was tender to pressure, the tenderness being most marked over the zygomatic arch.

A simple mastoid operation was done, showing the mastoid antrum and cells to be full of pus and granulations. The cells continued forward into the root of the zygoma. A short distance into the zygoma the cells seemed to stop. But on further investigation it was found that what appeared to be

compact bone was merely a thin bony septum, on the other side of which was a large cavity embracing the entire interior of the zygomatic process, which was converted into a hollow cylinder filled with pus with a perforation on its inner surface.

The skin incision was continued forward above the auricle to the hairline at the temple, through the temporal muscle and periosteum, and the periosteum was elevated from the squamous portion of the temporal bone, revealing a collection of pus in the temporal fossa, containing about two drams. The temporal muscle was very much thickened and edematous. After removing as much of the zygoma as was diseased the fibers of the temporal muscle and the anterior portion of the skin incision were sutured, and a gauze drain placed in the temporal fossa leading back into the mastoid wound. This drain was made shorter at each succeeding dressing, and after about a week and a half was dispensed with altogether.

On the day following the operation the temperature was 103° F. It gradually dropped, and in a few days was normal. Recovery was without incident. A culture of the pus from the abscess showed streptococci.

CASE 2.—Andrew T., a car cleaner, 30 years of age, was admitted to Dr. Berens' service at the Manhattan Eye, Ear and Thoat Hospital, on January 12, 1911, with a history of having had pain in the right ear beginning on November 24, 1910, followed by a bloody and then a purulent discharge, with persistence of the pain. On examination there was found a perforation in the upper posterior quadrant of the membrana tympani, with a canal full of pus. There was some sagging of the posterior superior canal wall and edema and tenderness over the mastoid.

A simple mastoid operation was done and a large perisinus abscess was found. The wound healed smoothly, until February 19th, five weeks after the operation, when he began to complain of a steady boring pain in the right temple and in front of the right ear. This pain persisted, and on March 8th swelling and tenderness were discovered in the temporal region, the swelling corresponding in size and shape to the temporal muscle.

The patient was reoperated by Dr. Berens, the incision being continued forward to the hairline, and the zygoma found to be necrotic and filled with pus, and a large collection of

pus found in the temporal fossa. Recovery was uninterrupted.

CASE 3.—Louis B., 18 years of age, a pocket-book maker, was admitted to Dr. Berens' service on November 26, 1910, with a history of pain in the right ear, four weeks before. It improved under irrgiation, and then grew worse again. At first he had discharge from the ear. A week before admission the discharge stopped and the pain became more pronounced, worse at night. On November 26th he was operated on by Dr. Berens. A simple mastoid operation was done. On December 4th the temperature rose to 102° F., pulse 108. He complained of pain in the right side of the head.

On December 7th, a blood examination showed a leucocytosis of 16,400, with 84 per cent of polynuclears. Blood culture was negative. There was edema over the temporal muscle, the size and shape of the swelling corresponding to that of the muscle.

On December 8, 1910, he was reoperated, and a deep temporal abscess was found, with an epidural abscess in the middle fossa, under the squama of the temporal bone. Recovery was uninterrupted.

Although deep temporal abscess is a clear cut and well defined picture, very little has been written about it. I was able to find very few references to it in the literature.

Macewen, in his "Pyogenic Diseases of the Brain and Spinal Cord," describes the condition in children, and reports three cases.

H. Gifford, of Omaha, reports two cases in the *Laryngoscope,* November, 1907.

Massier reported two cases in *La Presse Oto-laryngologique Belge,* January, 1903.

Raoult reported one case in *La Revue hebdom. laryngol.,* 1902.

Müller reported a case in the *Fortschr. d. Med.,* August 1, 1904.

Antonelli reported a case in *Rec. d'ophth.,* May, 1904.

616 Madison Avenue.

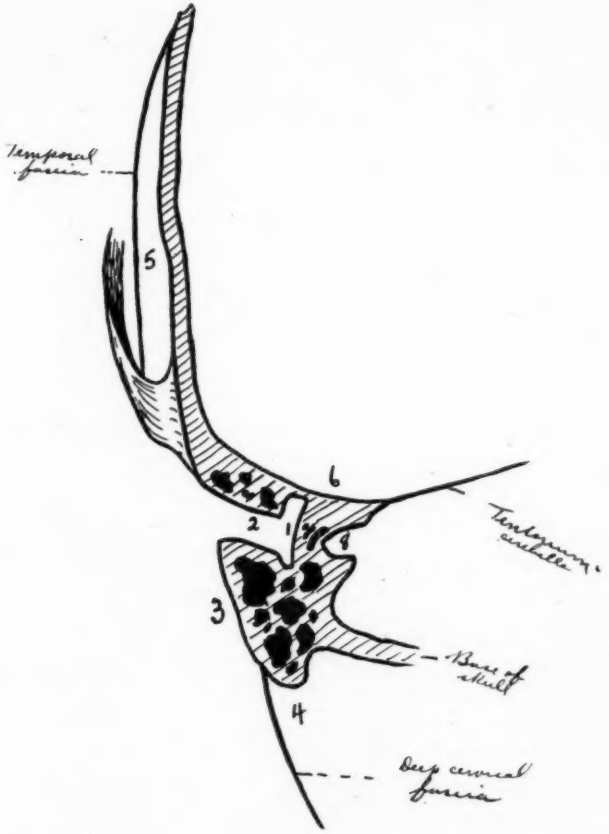

FIGURE 1.

Possible locations of perforations:

1. Through middle ear.
2. Through external auditory canal.
3. Through outer surface of mastoid.
4. Bezold's perforation.
5. Under temporal fascia.
6. Into middle fossa.
7. Into posterior fossa.
8. Through internal auditory meatus.

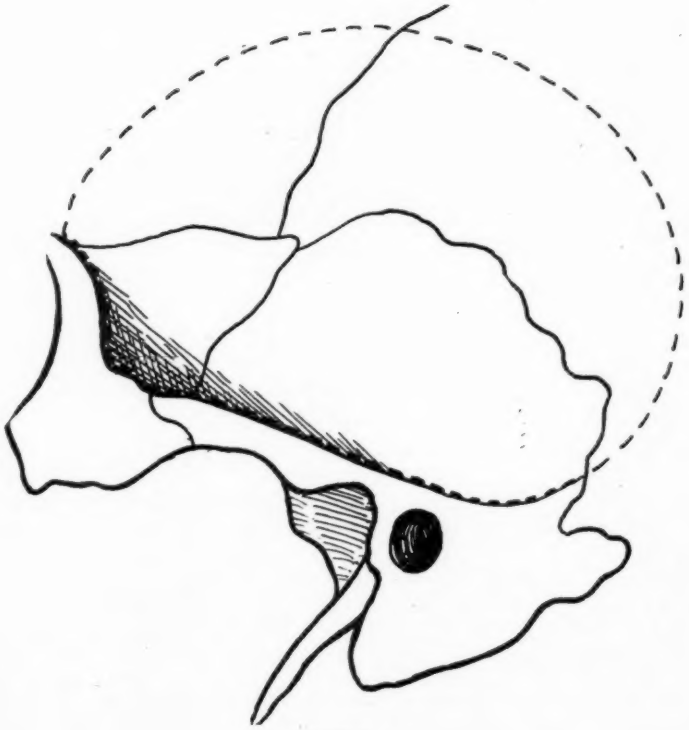

FIGURE 2.

Dotted line shows attachment of temporal fascia.

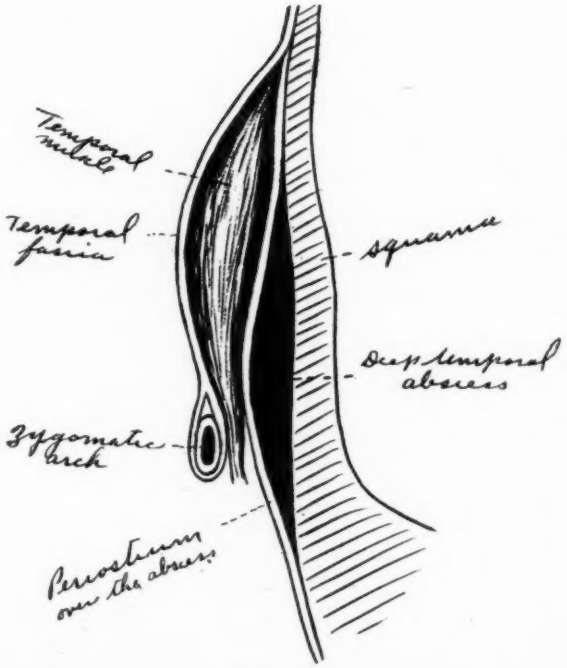

FIGURE 3.

Coronal section through temporal bone, showing situation of deep temporal abscess.

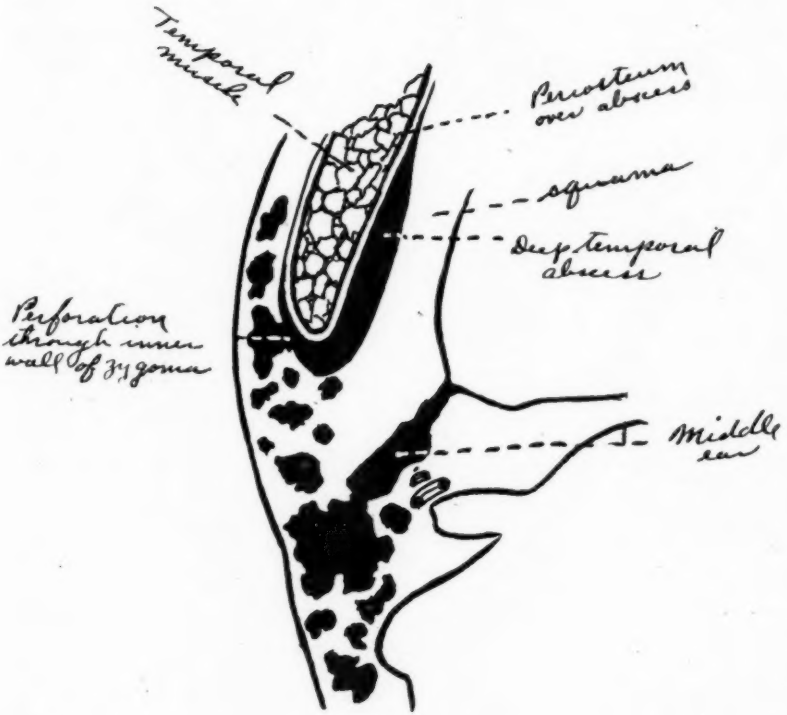

FIGURE 4.

Horizontal section of temporal bone, looking down from above, showing manner in which pus finds its way from perforation in zygomatic arch to outer surface of squama.

FIGURE 5.

Deformity in deep temporal abscess.

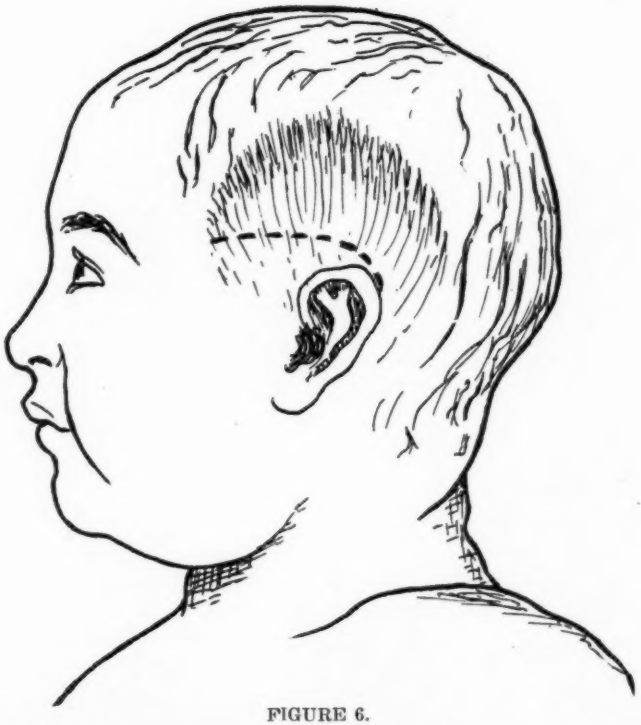

FIGURE 6.

Lateral view of deformity in deep temporal abscess. Dotted line shows extension forward of mastoid incision to edge of hair line.

VIII.

STREPTOCOCCUS INFECTION AND IMMUNITY.*

From the Laboratory of the Manhattan Eye, Ear and Throat Hospital.

By L. W. Strong, M. D.,

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The streptococcus is by far the most interesting of the bacteria of acute infections, because it is both the most common and the most virulent. Its interest in otology lies in the fact that it greatly predominates in the causation of acute otitis media, mastoiditis, sinusitis, and jugular thrombosis.

Other organisms which may produce these conditions are the pneumococcus and Friedländer's bacillus, but these are relatively rare; while one variety of the streptococcus, i. e., the streptococcus mucosus capsulatus, which is found not infrequently, forms a significant morphologic link between these three groups.

The explanation of this frequency is indeed its other character, namely, its virulence, or its capacity of attaining high virulence or invasiveness. This becomes most marked experimentally through animal passage, or in the cases of infection from one person to another, as at operation or autopsy, but also from the eclamptic course of an infection from a simple cut or abrasion, where an organism of the skin attains an irresistible virulence.

It will be of interest then to review the work which has been done on this organism and to consider the present status of methods of combating it.

The first and the classical investigation of the pathology of streptococcus infections was that of Bordet. He used a streptococcus whose virulence had been heightened by Marmorek so that it killed rabbits in a dose of a fraction of a millionth of a cc. This was injected intraperitoneally in guinea pigs, and the process watched by puncturing the abdominal

*Read before the Section on Otology, New York Academy of Medicine, October 13, 1911.

wall and withdrawing a small amount of the exudate every few minutes.

I. FATAL DOSE.

There first occurs the phagocytosis of Metchnikoff which regularly follows the introduction of bacteria intraperitoneally. These are the large mononuclear cells. Then come the polynuclear cells which begin to take up a few streptococci, but there are many not engulfed which are the most resistant ones.

The cells become infinitely more numerous than necessary to take up all the streptococci, but the resistant ones have a capsule or areola around them.

In 2 to 3 hours these increase so that in 6 to 8 hours we find a large number of cells and of cocci, but the phagocytes are empty. They are not dead or paralyzed, as may be seen by injecting some nonvirulent bacillus, as proteus, which will be engulfed. The streptococci then secrete a substance which, although it does not inhibit the influx of phagocytes, prevents phagocytosis. The streptococci thus exercise a negative chemotaxis.

II. NONFATAL DOSE.

Rapid and complete phagocytosis occurs. If the number of active phagocytes is increased by previous injection of bouillon two times m. l. d. is overcome. The bacteria do not have time to adapt themselves and increase their resistance.

Bordet concluded that there was no other factor than phagocytosis in the recovery of an animal from a streptococcus infection.

The fluid in which streptococci are grown contains no antiphagocytic body; this is associated only with living streptococci, and in my opinion only in living tissues.

The momentary arrest of infection and apparent cure are due to intervention of complete phagocytosis and the consequent disappearance of free bacteria.

But the more resistant cocci live and reproduce a more virulent strain. This ultimately grows free in the abdomen and overwhelms the pig.

Bordet next observed that an infinitely smaller quantity of his virulent culture would produce death in rabbits than was required for guinea pigs.

Why do pigs resist? Normal sera are favorable for the growth of streptococci; there is no bactericidal power, and they also grow in the exudate of infected pigs.

The pig serum has no more antiseptic properties than rabbits, but pig leucocytes are much less sensitive to the repelling action of the streptococci.

As much as .5 cc. of a young culture of streptococcus is rapidly taken up in the exudate of a pig, while in a rabbit the same amount is not much engulfed.

This difference in the reaction of the leucocyte explains clearly the difference in the evolution of the disease in the two animals.

Experiments in vitro with leucocytes cannot be accepted offhand without many reservations as necessarily corresponding to the conditions of similar experiments in the animal body.

This theory of phagocytosis is met by the undeniable fact which Bordet himself brings out, namely, that antistreptococcus sera are at least preventive in their action. If then a serum may have some property which increases resistance, it is plain that phagocytosis alone is insufficient to account for natural immunity. Furthermore, the equally incontrovertible fact that animals may be immunized by streptococcus inoculations can be explained only by the intervention of some body in the serum; it is inconceivable that the inoculations act directly on all the leucocytes in the circulation or in the leucocytic reservoirs.

All investigators, Metchnikoff and Bordet included, have noted the favoring influence of antisera on phagocytosis. Denys and Leclef and Neufeld and Rimpau corroborate this for the streptococci and call the substance bacteriotropin. Even earlier, Wright employed the term opsonin. This is considered an individual substance by some, and by others is regarded as identical with sensitizer.

The opsonic action of normal or of specific sera is in the main a combined action of sensitizers and alexin. In the specific serum the action is increased and brought about chiefly by the sensitizers.

This review of the history and cause of the streptococcus infection has been for the purpose of discussing the various methods of its specific treatment and what may be hoped or expected from them.

All specific treatment depends upon one of three things—antitoxic, bactericidal, or opsonic activity.

First—Antitoxic: The streptococcus belongs to that group of bacteria which produce endotoxins, that is, those bound up in the vegetable cell and only separable from it at death of the cell. Indeed, according to Hiss' views, the real toxins are possibly only developed after partial digestion by the combined sensitizer complement.

Aside from the relatively unimportant hemolysins and leucocidins produced by the streptococcus, its real toxin is unknown, and it is certainly a very striking circumstance that an organism which can be so rapidly fatal should have no known specific toxin.

It may fairly be hoped that ultimately its toxicity may be better understood and possibly antagonized, but at present there are no attempts at such therapy.

Second—Bacteriolytic: It is generally held that normal sera are at least to some degree bactericidal or bacteriolytic, and the question at once arises whether this property may be partly responsible for the normal immunity of the guinea pig, and whether the bactericidal activity may not be increased by immunization and become a means of combating infection.

The experiments to demonstrate the bactericidal activity of normal sera or streptococci have never seemed conclusive to me and have seemed open to certain objections. Thus the method has been to place streptococci in fresh serum, and after a certain exposure to plate the mixture on agar and to count the colonies which develop. But streptococci grow poorly on agar at best, and there is a considerable margin of error in counting the extremely minute and colorless colonies which develop.

Aside from this, a far more serious objection is that the streptococcus being in chains of individuals, the resistance of a single coccus in a chain would cause a colony to develop so that the destruction of the other individuals would be masked.

To avoid these errors and to see what actually occurred in the living body, that is, in the plasma rather than the serum, I have exposed living virulent and avirulent streptococci in permeable celloidin capsules in the peritoneal cavity of guinea pigs for one to four days. At the end of that time the strep-

tococci were found alive and with undiminished virulence, and sometimes it was obvious that they had multiplied tremendously. That the capsules were permeable was obvious from the fact that in some instances a few leucocytes had penetrated them.

From these repeated experiments I conclude that the guinea pig plasma, though the animal is normally highly immune to the streptococcus, is not bactericidal.

A large number of experiments, also, in which virulent and avirulent streptococci have been planted upon fresh guinea pig serum, have led me to conclude that while the serum affords a poor culture medium it is not bactericidal for the streptococcus.

From these data it appears that bacteriolysis by the plasma is not a factor in streptococcus immunity.

The third method of specific treatment, then, remains—phagocytosis; and it is in efforts to heighten this that all forms of specific treatment resolve themselves.

There are three such methods in vogue, namely, vaccines, immune sera, and leucocytic extracts.

The vaccine method devised by Wright seeks to produce an active immunity by the production of opsonins by the tissue cells locally at the site of inoculation.

Some of the theoretical objections of this method are, firstly, the organism is already undergoing stimulation to the production of such opsonins through the disease process itself, and, secondly, it has been questioned if in reality there is such local production.

Hektoen has shown recently that in the case of albuminoid bodies they are not developed in the subcutaneous tissues.

Secondly, the use of immune sera. At the meeting of the Pharmaceutical Section of the American Medical Association, 1910, reported in the *Journal of the American Medical Association*, No. 54, p. 257, Weaver and Tunnicliffe stated that no antistreptococcus serum on the market had been found to be either antitoxic, bacteriolytic, or opsonic.

In the September number of the *Journal of Infectious Diseases*, however, the same authors state that they have continued their work, using a modified Wright technic, and they conclude that antistreptococcus sera are opsonic; that this property disappears upon standing, and may be reactivated by fresh human or guinea pig sera.

These sera are, moreover, known to be protective when introduced into a test animal together with streptococci.

Thirdly, leucocytic extracts: The work of Hiss is based upon the conception of the leucocyte as the ultimate source of bacteriotropic or antiendotoxic bodies which are not normally given up to the plasma.

Although not normally circulating in the plasma, he relies upon their absorption into the circulation and their subsequent neutralization of the bacterial poisons.

Rabbits treated with these extracts of rabbit leucocytes after injection with virulent streptococci showed a remarkable percentage of recovery.

One must here assume that the extracts actually represent the bacteriotropic or antiendotoxic bodies, that they are fairly stable, and that they are in fact absorbed unchanged, and that the rabbit extracts are equally available for man.

We know that the system quickly accommodates itself to the introduction of foreign albumens by the production of antibodies.

Moreover, since leucocytic extracts rely on antiendotoxic properties, they can be regarded only as secondary agencies in combating infection where phagocytosis is the natural mechanism.

It will be noted that the vaccine method seeks to secure active immunity, while sera and leucocytic extracts, affording passive immunity, aim to introduce bacteriotropins direct and preformed.

The objection to the general principle of opsonic treatment of streptococcus infection, which applies equally to vaccines and antisera, lies in the fact that the system itself is under violent stimulation to the production of these antibodies, and, according to Ehrlich's general law, whenever the tissues receive such stimulation the tendency is to the production of the bodies in excess.

This may be a positive disadvantage by producing complement deviation, using up the complement necessary for completion of the opsonic effect.

It is undeniably true that antistreptococcus serum may protect animals when introduced at the same time with virulent streptococcus, but this unfortunately is a very different matter from a real infection, for in the very first stages there are

no specific opsonins ready to meet the attack of the bacteria. When these are introduced, as in the case of a protective serum, the organism has at hand normal alexin or complement, and this plus the preformed opsonins of the protective serum enables the leucocytes to engulf the bacteria. But after the streptococci have once begun to multiply, the burden is shifted off from the opsonins, which now, if anything, are in excess, and the complement is the factor to be preserved.

Bordet's view is that the conception that under immunization an animal forms an appropriate sensitizer capable of causing the bacterium it affects to absorb complement, does not necessarily imply curative or protective properties for normal animals. We may simply say that the function of sensitizers in protecting animals varies with the bacterium. Certain ones are easily destroyed in fixing alexin, others resist better, and others doubtless absorb it with impunity. In animals invaded by bacteria death is scarcely due to lack of alexin, but to inability of phagocytes to ingest the bacteria; and even supposing alexin protection to be the essential factor, death would be due not to such lack, but to lack of utilizing or absorbing alexin.

We should scarcely expect in treating human bacterial infections that, as Wassermann has suggested, the administration of certain normal sera or alexins in addition to specific could be recommended, since the alexins from foreign species affect not only bacteria, but body cells; and, moreover, the animal would soon protect itself from such injections by forming antialexins.

Weaver and Tunnicliffe state that by actual experiments they have several times found fresh serum of an individual with a streptococcus infection able to reactivate antistreptococcus horse serum, even though the patient's blood contained less opsonin than normal.

We have, then, two possibilities and two contrary views of the injection of immune bodies: either that they may be ineffectual by being nonprotective, or even harmful by complement deviation; and, on the other hand, that they may be useful when the patient has a deficiency of opsonin and an excess of complement.

This, it seems to me, is the weakness and the unscientific aspect of all specific treatment as at present conducted. This

may, however, be obviated and should be controlled by some technic which would tell us the approximate relationship of opsonin and complement, and in the individual case, from hour to hour.

Wright's opsonic technic is an effort in this direction, but unfortunately, in my hands at least, unavailing.

Weaver and Tunnicliffe draw their conclusions as to the opsonic effect of antistreptococcus serum not only from the modification of Wright's technic, but from the method of dilution to opsonic extinction.

They find the two methods to give fairly uniform results, which gives much more weight to their conclusions than would the Wright method alone, which has been found faulty by so many observers.

For myself I have found it lacking in that I have very numerous observations upon streptococci freshly isolated from lethal processes which were very readily phagocyted without any trace of serum.

To obviate this I have employed streptococci washed from the peritoneal cavity of animals dying from streptococcus injections. It is only then that they are nonphagocytable.

Even when these were employed I have been unable to demonstrate by Wright's method that normal guinea pig serum had greater opsonic activity than rabbit or human, which it should have if its immunity is opsonic.

However, I have accomplished the same purpose by demonstrating that normal guinea pig serum, like antistreptococcus sera in general, was protective to mice when introduced with virulent streptococci, and caused a very rapid disappearance of the cocci from the abdomen.

These may be found phagocyted; and markedly not in the leucocytes, of which there are few at this early stage, but in the endothelial cells of the lymphatics of the mesentery.

We know by staining reaction that the streptococcus may be alive after ingestion and may live several days and later reinfect. In addition to negative chemotaxis and phagocytosis, then, there must be lysis in the cell.

Lamar, Noguchi and others have shown that the lytic bodies expressed from the leucocytes are in the nature of soaps and fatty acids. But this intracellular lysis is more probably an enzyme activity in the nature of digestion.

Lamar's conclusion, that for pneumococcus antiserum, bacteriolysis in the presence of soaps is the method of protection, seems to be a special phenomenon not associated with the streptococcus where opsonic activity appears the factor.

Denys, Leclef, Neufeld, and Töpfer observed an increased phagocytosis in vitro with immune serum. Metchnikoff regards this as a stimulant to phagocytosis, which Neufeld and Töpfer call bacteriotropic (opsonic).

Against Aronson's and Marmorek's sera, which are made from streptococcus with heightened virulence for animals, is the fact that this modified animal culture results in a strain which has no high virulence for man.

Aronson now uses a serum half from animal virulent streptococcus and half from horses immunized directly with a human virulent strain.

The value of a serum is estimated from its protective power to mice. There is no unanimity in the reports on the use of these sera.

The high specificity of the streptococcus and the fact that the amboceptor is also purely specific, may make an animal serum unavailing.

If any immune body be used in specific therapy, it must be administered at the earliest possible moment, intravenously, and in large dosage.

IX.

EXPERIMENTS WITH THE AUTOLYTIC SOLUTIONS IN THE TREATMENT OF INOPERABLE CANCER OF THE THROAT, NECK AND FACE.*

By Joseph C. Beck, M. D.,

Chicago.

About two years ago, by way of personal communication with Dr. Carl Wagner, of Chicago, who had just returned from studies abroad, I first learned of the experiment that had been carried on by Dr. Fichera, of Rome, in the treatment of inoperable carcinomas and his good results. I did not pay any more attention to the subject until last July (1911), when Dr. Emil Beck returned from a similar trip and described to me in detail what he had learned at Dr. Fichera's clinic. His great enthusiasm impelled me to associate myself with him in carrying on some experiments on cases of advanced carcinoma. I desire at this time to report on the work thus far done on my cases. Permit me, first, to state the theory upon which this treatment is based. I should, in justice to the author of this theory, quote verbatim from the excellent article, published, but neither time nor space will permit me to do so.

THEORY.

Assuming Cohnheim's theory, that at birth there are nests of embryonal epithelial cells distributed all over the body, that these cells always remain quiescent, and only through some form of irritant, either mechanical, chemical or through the agency of some organism, they begin to grow. This growth, however, does not, as a rule, take place until near the fortieth year of life. It is further assumed that at birth there is a substance in the blood which is inimical to the growth of these stray cell nests, and as long as it is present in sufficient quantity and quality, even though the mechanical, chemical

*Read before the Southern Section of the American Laryngological, Rhinological and Otological Society, February, 1912.

and organic irritation be manifest, carcinoma will not develop. It is only when this substance is diminished in quality and quantity, or entirely lost, that these irritants enable the stray epithelial cell nest to grow. The usual time of life when the loss of activity of this substance in the blood occurs is at about forty years of age. By supplying artificially, that is, by injecting subcutaneously, a solution that contains this substance, which prevents the development of carcinoma, prophylaxis is possible. This substance is contained in all embryonal tissues especially, but is also contained in the carcinoma cells. If, therefore, carcinoma has developed, then the further growth of it can be checked, and what has already developed brought to nonactivity, by injecting these solutions, which are known as "autolytic." Of course, these injections must be continued at definite intervals during the remainder of the individual's life.

Only inoperable cases were taken for these experiments at first, so that the early reports of Fichera were not as good as later, when better material was at his disposal.

The many steps in the development of these experiments are all very interesting, but too lengthy and not essential.

The preparation of the autolytic solution may be obtained in two ways, namely:

1. In case that we employ the carcinoma itself, it is essential that it is obtained absolutely sterile. Therefore, metastatic glands are best or a tumor not accessible to the outer world. The tumor is, therefore, removed under the strictest aseptic condition, and is cut up into small cubes, 1 centimeter square. These are placed into a jar of sterile normal salt solution. About twenty times in bulk of salt solution is employed to the tumor cubes—that is, for each centimeter of tumor twenty cubic centimeters of salt solution are taken. To this are added thymol crystals, twenty grains to the pint of solution. On top of this is poured a quantity of sterile olive or any other oil, so that all is covered and prevented from too easy contamination. The jar is carefully closed and placed in the incubator at a temperature of 37° C. for sixty days, when it is ready for use.

2. In case we employ a fetus, it is best to obtain one at about three to six months of age. The great difficulty is in getting the fetus without contamination during its expulsion from the uterus. The entire fetus is made use of with the exception of

the scalp and brains, which are very carefully removed before the rest of the body is cut up in very small cubes, as when the tumor itself is employed. The remainder of the technic in the preparation of the solution is also the same.

In order to be certain that the solution is sterile, a few drops should be withdrawn with a sterile pipette and plated. If there is no growth, two or three cc. of the solution should be withdrawn and injected subcutaneously near the seat of the carcinoma, if possible. This is repeated two or three times a week, and continued until the growth has disappeared.

Besides the clinical picture, the microscope is used as a control. Fichera has sent us some specimens of carcinoma which he excised at different intervals of the treatment, and they certainly show the good effect from the injections. The first specimen showed almost a pure epithelial growth, which was excised just before the first injection. The second specimen, about three weeks after injections, showed marked diminution of the epithelial structures and a great increase of connective tissue changes. The third slide has but very few cells of epithelium. At the same time the clinical report showed that the growth was practically gone and the patient in good condition.

CASE REPORTS.

CASE 1.—Mrs. F., 37 years old. Developed a carcinoma of the breast. This was removed radically, including the axillary lymph glands, by Dr. Emil Beck. A few months later there was a recurrence in the scar and some diffuse nodules presented over the pectoral area. Another operation was performed for these recurrences. About two months later the patient returned with a number of enlarged glands of the neck, extending from the clavicle up to the posterior triangle of the neck. These were quite movable, although they were causing a great deal of pain, radiating into the arm. An operation was decided upon and the case was referred to me for further care. I removed a number of enlarged glands which were matted together, and it was necessary to remove the internal jugular vein with the mass, owing to its firm adhesion. The operation was performed under the strictest aseptic precautions, and these glands cut up in the manner described above, as also the further technic in preparing the autolytic solutions

which were to be employed in this case. The wound healed by primary intention and we began the injection of from two to three cm. twice weekly, about seven weeks after operation. The patient had some small nodules on the chest, which were not disturbed at the time of the operation, but a small piece was taken for microscopic control. It was a typical medullary carcinoma, containing very little fibrous stroma, but a great deal of broken-down cell tissue. The patient was free from pain and continued to improve in her general condition. After three months we found that these nodules on the chest were getting harder and there was no recurrence at the site of the neck wound. The treatment is being continued, and I shall in the very near future remove a piece of tissue from these nodules for control microscopic change.

CASE 2.—Mr. J., 52 years old. Has had a sore on the side of his face, just in front of the parotid gland, for the past six months. It had been treated by local measures, including X-ray. No history of lues or tuberculosis was present. I removed a small piece for microscopic examination and found it to be a very active epithelioma. No glandular involvement. Operation was decided upon and a thorough removal of the infiltrated mass wide of the affected margins. It was found necessary to remove the following: Parotid gland, part of malar bone, external orbital wall very near to the optic foramen but not wounding the orbital periosteum, part of the masseter muscles and the coronoid process of the lower jaw. This extensive dissection appeared necessary from the apparent pathologic involvement of the epithelioma. At one place in the region of the antrum there appeared a suspicious area which I carefully curetted without going into the antrum, which I very much wanted to avoid doing. This proved the failure of the operation, because from this suspicious area the antrum became infected, and once in this region the tumor grew more rapidly and spread over the wound of the cheek.

Since we had no metastases in this case, and the tumor mass removed could not be obtained sterile, I decided to employ the autolytic solution produced from a fetus. The patient has had too few injections thus far to make any comments upon the case as to the influence, but I wish to place it on record for future report.

CASE 3.—Mr. K., 55 years old. For the past four months

has been hoarse and coughs a great deal. During the last three weeks he noticed two large and some small lumps on the outside of the neck. Diagnosis of carcinoma of the larnyx with secondary infection of the glands of the neck was made by exclusion. I decided to operate in order to obtain the metastatic glands for the purpose of making an autolytic solution for injections; also to place a tracheal canula in position, for it was evident that it would soon be necessary. A transhyoidal incision was made, the glands removed under the strictest aseptic conditions, and then the tracheotomy performed. The patient made an uneventful recovery and was getting along well under the use of these injections. While he did not gain much in flesh, he was less toxic and ate well. During the severe cold weather he would come to the hospital for treatment, and following a wait for the street car one evening, he came home very ill. He developed an acute bronchitis, which was soon followed by pneumonia and death. There was positively no increase in the laryngeal growth during about three months of treatment. I was very much interested in the course of the neoplasm, in that it remained stationary and no further metastases were in evidence. Of course, a postmortem examination would be necessary to prove that point, but that was not permitted.

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X.

CONTRIBUTION TO THE PATHOLOGY AND TREATMENT OF OTOSCLEROSIS.

By Joseph C. Beck, M. D.,

Chicago.

In studying the various pathologic processes in bones of the human body, I have noted a great similarity between what is known as osteomalacia and what otologists call spongification of the bony capsule of the labyrinth. In this connection I became interested in the work done by Bossi and his treatment with adrenalin chlorid solution hypodermatically. About two years ago I saw a case of osteomalacia in the care of Dr. Carl Beck, a young lady about twenty years of age, who gave a history of having had a number of spontaneous fractures which were brought about by the slightest exertion; in fact, in bed, by the action of a severe muscular spasm. These fractures were set and in some cases opened and wired, but they healed very slowly or not at all. After about six months of this sort of treatment, C. Beck began the injection of adrenalin solution subcutaneously, beginning with one drop and increasing by one drop daily until ten or fifteen minims were used, when the dose was again reduced, probably to two to three drops. The treatment was continued for several months, the patient improving in general health while fractures did not recur. The patient was able to walk about with the aid of crutches. About this time Dr. Carl Beck made his report on this and another similar case, with a complete review of the literature.

As already stated, I had been much impressed by the similarity in the bony changes of osteomalacia and otosclerosis, so I decided to experiment with a number of cases. It is stated that osteomalacia is a disease process for which the improper or disturbed function of the organs of internal secretion is responsible, the adrenal gland in particular. Hence, by the artificial administration of the adrenal solution the process is brought to a standstill, as shown clinically. I thought that

possibly the same cause was responsible for the development of otosclerosis, and consequently placed a number of cases upon this experimental treatment. I selected three groups of cases, eleven in all.

The first group consisted of those which have practically run their course and are sclerosed, with marked involvement of the endorgan of hearing. One would scarcely expect any results so far as the hearing is concerned. While they were practically deaf, they had severe symptoms, as dizziness, vertigo, and marked subjective noises. They responded easily to the vestibular reaction by turning; inasmuch as they had severe tinnitus, the presumption was that some other part of the nervous system may be responsible for this (H. Neumann).

The second group of cases was composed of those of more recent origin, who had still some useful hearing, at least in one ear.

The third group constituted those of an indefinite type. They had fair hearing, with clear histories of otosclerosis, but with considerable evidence of tubal and middle ear inflammation. In one case there was suppuration in both ears, completely cured, but with scars in evidence about the drum.

The histories of all these cases were carefully prepared and in each instance a family tree slightly modified from those presented by Gray (Glasgow) was made (Fig. 1). The tests of hearing were recorded, the conversational tone instead of forks being taken as a standard for testing the progress.

The treatment was carried out as follows: Patients came usually about one hour before the evening meal. After being seated for five minutes, so as to get the normal blood pressure, a von Stein apparatus was placed on the radial artery and the blood pressure recorded in a book. Now, one minim of an adrenalin solution, 1-1000, is injected hypodermatically into the arm. After three minutes the blood pressure is again taken and recorded. (Not until three minims are injected is there a change noted, as a rule,) Granting that the blood pressure at the beginning was 146, it now shows an increase to 147. Five minutes later the blood pressure was again taken; at this time it will be at its height, 149 for instance. Five minutes later, or ten minutes from the time that the injection was made, the blood pressure is again taken, and ordinarily it is found that it has gone back to the normal—146. On the next day

this procedure is repeated, except that the dosage is increased one drop; this is continued for successive days, so on the tenth day there will be ten drops of adrenalin solution used. The figures will be something like this: 146, 149, 153, 146. If now, on the fifteenth day, the blood pressure goes after three minutes to 155 or thereabouts, and after five minutes to the highest point, 165, it indicates that we are reaching the physiologic limits, and we will find that the last blood pressure measured will not go back to 146, but will remain for some time above 150. We now stop injecting for a few days, and then begin again with one minim and increase. However, we never go again in this case as high as fifteen minims, but stay at ten minims, and continue as long as the blood pressure returns to normal. It, therefore, must be made a rule that the blood pressure must be the same ten minutes after the injection as it was before the adrenalin was injected.

RESULTS.

In the four cases in which there was no conversational tone heard, the vestibular irritation which was present was greatly reduced, and in one patient the "terrible" noises ceased. The hearing was not influenced in the slightest. Of the three cases in the second group, all three sisters, the eldest twenty-six years old, the youngest sixteen years old, who still have fair hearing in one ear, all improved in their hearing according to the reports of their employers and people about them. In the third group of the mixed or indefinite cases, we have noted some interesting observations. I placed them first on the adrenalin alone; then I added other ductless gland extracts, so that one case (a male) received adrenalin, thyroid, thymus. The second case (a male) received adrenalin, thyroid, thymus, spleen, pancreas, testicle. The third case (a female), adrenalin, thyroid, thymus, pituitrin and ovary. The fourth case (a male), all the above mentioned, with the exception of the ovary, and, in addition, parotid.

It must not be assumed that these organ extracts were employed indiscriminately and all at once. They were administered usually alone or two together, and the result recorded.

After several months' experimentation on these last four cases we found certain definite general symptoms, as acceleration of pulse, giddiness and vasomotor changes, as flushes, etc.,

but the analysis of these will require a great deal of time and study, and they do not appear to have any influence on the symptoms referable to the ear, that is, the deafness and disagreeable noises.

It is not intended to bring this report as a cure in this most deplorable condition, but only a report on some experiments, hoping that something has been contributed that may start some new thought which eventually will be of benefit in otosclerosis and allied affections.

I desire to express my appreciation to our pathologist, Dr. Clara Moore, who so diligently observed and recorded these cases, and who did most of this work under my direction.

In conclusion, I wish to say that only private cases are suitable for such work, because they can be relied upon. Again, in all the experiments we have not had one single instance of trouble or complication. The duration of the treatment was from three to six months, and after that they were under observation, say once a week, without treatment.

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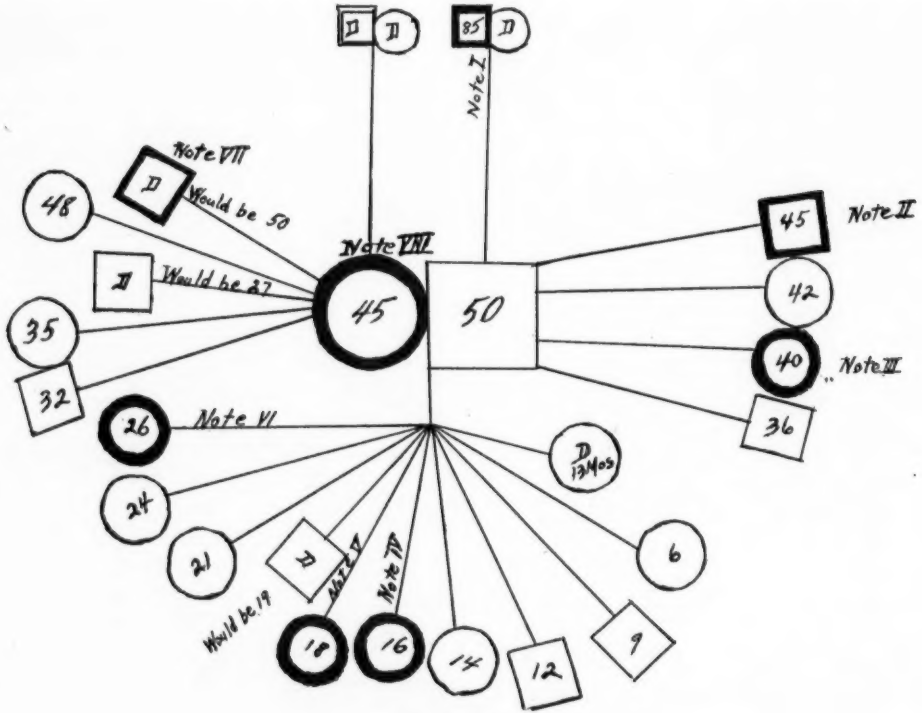
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XI.

CONTRIBUTION TO THE ETIOLOGY, PATHOLOGY AND TREATMENT OF ATROPHIC RHINITIS.

By Joseph C. Beck, M. D.,

Chicago.

In February, 1907, I presented a paper before the Middle Section of this Association on this subject, in which I brought out my results in the X-ray examination of atrophic rhinitis, showing that the sinuses appeared to be involved, and in the discussion, Coakley showed some radiograms verifying the assumption, but he believed that the sinuses were smaller than normal, but not necessarily the seat of disease. It was and still is my opinion that the sinuses, especially the ethmoidal labyrinth, are the seat of pathologic changes. It is not possible to tell whether these cells are infected primarily, and the atrophic changes are secondary, or vice versa. About the same time Casselberry published his article on the subject of ethmoid operation for the improvement of this condition. I myself have had gratifying results with this method of treatments. I also presented at the meeting a résumé of the various treatments in vogue, and mentioned my results with vaccines, Bier's hyperemic treatment, and paraffin injections into the inferior turbinate.

Since that time I have been investigating the etiology, pathology and treatment, which I desire at this time to place on record.

In looking over the histories of my private patients suffering from atrophic rhinitis, I was somewhat surprised at the predominance of the female sex. I always knew that most of my cases were in young girls, but not that the percentage was so great. Out of 57 cases there were 46 in females, and 11 males. Seventy-five per cent were in young ladies between the ages of nine and twenty-four years, and most of these between sixteen and twenty. I began to study the histories of these cases

and found that an anemia was very prevalent, and in several instances it was stated that the patient was treated for the green sickness or chlorosis, and also that there had been menstrual disturbances. It then occurred to me that perhaps there is some blood change that could be demonstrated in these patients, and therefore I selected a few cases to determine this point. A thorough blood examination was made in every case, but aside from a relative anemia which could be accounted for from this constant absorption of the purulent and fetid material in the nose, nothing of a blood change was noted. I then began the pathologic study of these cases, beginning with the histologic and chemical analysis of the crusts, including the bacteriology of the secretion, then histologic examination of the middle turbinated and ethmoid cells. One very striking feature in the composition of the crusts was the marked amount of fibrin present. This was found in all the cases, and led me to further investigation, namely, the examination of the blood for the quantitative fibrin test. We followed the method as suggested by Simon, and came to some constant results. At first we tested a number of normals as controls, and found that the quantity of fibrin in 10 cc. of blood was about one-half to the same amount of blood from an atrophic rhinitis case. This suggested to me the experiments with agents that would influence the fibrin formation in the blood. We found that the desired effect was attained by the use of inhalation of nascent oxygen and the interdiction of table salt as far as possible; in other words, a salt-free diet; as well as of foodstuffs that contain calcium salts. After several weeks of this line of treatment, we reexamined the blood for the quantity of fibrin and found at least a reduction, if not a return to the normal. From these experiments, although too few cases and too short a time has yet elapsed, I believe that the blood changes play an important part in the causation of this disease.

In the pathologic investigation of the middle turbinals and ethmoid cells I found the same changes in the mucosa as have been demonstrated in the inferior turbinals, only not so marked, namely, a metaplasia of the epithelium into a sort of a dermal layer, showing evidences of desquamation. The mucous glands are much altered or atrophied with areas of round cell infiltration. The bone does not appear very different from a turbinal in suppurative sinus disease. The same is true of the

examination of the ethmoidal portions removed. Logan Turner and his assistants have recently studied this subject and have demonstrated practically the same changes in the turbinals.

The bacteria constitute a very rich flora of many types of organisms, but the large bacillus, known as the bacillus fetidus ozenæ, and the staphylococci predominate.

About a year or so ago I learned of the successful method of implanting or injecting paraffin below the mucoperichondrium and periosteum in the atrophied nasal chamber. These injections should be in line with the atrophied inferior turbinated body, and in the shape of a ridge or deflection. Wileminski first suggested this method of treatment abroad, and Kahn, of Chicago, in this country.

I had found in my early experience with paraffin injections into the inferior turbinated body that in most of the cases not very much could be injected without having it perforate and escape into the free nasal chamber. This, of course, could be explained from the pathology of the inferior turbinate, which is transformed into scar tissue, covered by a thick layer of diseased epithelium. I wished to satisfy myself of the condition of the mucoperichondrium and periosteum of the septum in these atrophic cases before injecting any paraffin, and to prevent what occurred when the inferior turbinate was injected. I therefore removed a piece of mucoperichondrium in a case of atrophic rhinitis in which there was a deflection of the septum and examined it microscopically. It is stated that atrophy seldom occurs when there is a deflection present, and this I have found to be the case. If the atrophy does exist in such a case, there is not so much difficulty in expelling the crusts. This is the reason for utilizing paraffin injection into the septum. The microscopic examination of this mucoperichondrium revealed that not alone was it not atrophied, but hypertrophied, especially the perichondrium. The epithelium showed some slight metaplastic changes. It occurred to me that these desquamative changes of the epithelium take place more readily when the nose is douched, and especially if ordinary water is used. This I found to be the case, because by withholding the water to a large extent and if used employing solutions containing some substance like ichthyol or, preferably, thigenol, desquamation was lessened. Oily products, as vaselin and sprays of the hydrocarbons, appeared to increase the putrefactive changes.

By reason of these investigations I have come to certain conclusions, based upon which is the treatment that I pursue, and which is about as follows:

For about two weeks, daily treatment in the office by mechanically removing the crusts, employing Bier's suction pump and applying to the entire surfaces, especially to the olfactory regions, a twenty-five per cent solution of thigenol in distilled water. The use of water douches and sprays is prohibited. A tank of oxygen is ordered for home use, and the patient instructed to breathe it in by way of the nose and out through the mouth from three to ten minutes night and morning. This breathing is to be done with considerable force and deeply, so as to reach every crevice and also distend the lungs properly. A culture is usually made on the first day of the treatment, and at the end of the week an autogenous vaccine which has been prepared, is employed in the usual way. If the improvement is apparent, then this line of treatment is continued, with instructions as to diet and hygiene, etc., but, as a rule, the removal of the middle turbinal and ethmoid curettage are suggested and performed. As soon as healing has taken place, the local and general treatment is continued until the mucous membrane has a much healthier appearance. Usually at the end of the second month of treatment the submucous dissection of the mucoperichondrium and periosteum is performed and a quantity of paraffin, with a melting point of 120° F., is placed into the pocket created and the small incision closed with a suture, or the lips of the wound coapted and a loose tampon put in to retain it. The size of this artificial ridge is to be judged carefully, so as not to overdistend the space and have a necrosis in consequence. Instead of incising and dissecting the membranes from the septum, a flat needle may be introduced as far back as possible between the bony and cartilaginous septum and the periosteum and perichondrium. Then a paraffin syringe is attached to it and injection into this space is made instead of laying it in. I have had the best results when I first dissected and put the paraffin in, because it is easy to perforate the membrane posteriorly and thus a complete failure result. In this manner I have been more satisfied with the management of cases of atrophic rhinitis, and am certain that the result of the treatment has been more satisfactory than I have found by other methods.

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NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of October 13, 1911.

Paper: Three Cases of Meningitis With Recovery After the Use of Urotropin and the Hiss Extract of Leucocytes.

By James Garfield Dwyer, M. D.,

New York.

DISCUSSION.

Dr. T. J. Harris said that he wished especially to congratulate Dr. Dwyer on the first two of the three cases reported by him. Nothing connected with meningitis has been of so much encouragement as the discovery of this Hiss extract of leucocytes, and of urotropin as used in lumbar puncture and also by mouth. He expressed his regret that Dr. McKernon was not present to report on three cases where he had used urotropin as Dr. Dwyer had used it; and he desired to emphasize Dr. McKernon's statement in regard to these cases, that if urotropin is to be used it should be given in large quantities, by lumbar puncture, not less than forty grains at the first introduction, to be repeated twice a day afterward until the indications for its discontinuance. They are using it at the Massachusetts Eye and Ear Infirmary as a routine procedure by the mouth, giving it in small doses, five grains two or three times a day, in all cases.

For the sake of those who had not been present when Dr. Dwyer read his previous paper, Dr. Harris said that he hoped Dr. Dwyer would refer briefly to his very successful experience with the Hiss extract. This is only one of a considerable number of cases that he has had, though it does not seem that either Dr. Dwyer or anyone else is yet prepared to state definitely where the Hiss extract should be used and where the urotropin. Presumably, no mistake would be made in employing both.

Dr. W. Sohier Bryant said that he has had some little experience with urotropin by the mouth, and had had no success with it. He has never used it in the spinal canal, though he had seen a case so treated last summer; the patient died. Dr. Flexner at the Harvey Lecture made some very interesting remarks in regard to this subject. He said that you cannot do anything with urotropin in meningitis, given by the mouth or through the skin. All agents must be applied directly in the spinal canal in order to produce the desired local effect. However, when there was trauma, the cerebrospinal serous sac acted more like other serous cavities and responded to general medication. A case now in the New York Eye and Ear Infirmary is like the first case described by Dr. Dwyer this evening—cerebrospinal meningitis with turbid spinal fluid, under pressure containing streptococci. This patient was operated on two weeks ago. He had a temperature of about 102°, pulse beats 40 to 50, was comatose, but could be roused. There was also rigidity of neck, and choked discs were present, symptoms rapidly becoming more alarming. It was a case of meningitis following acute ear trouble. A decompression operation was done. The case appears to be perfectly well, except for the wound, which has not healed yet.

Dr. Dwyer said that he had used the urotropin at Dr. McKernon's suggestion. A Baltimore physician suggested it to him, and in the case referred to by Dr. Harris, a case of cerebellar abscess and purulent invasion, he advised that 100 grains of urotropin be given daily, and the patient recovered consciousness after the first dose, although he had been unconscious for six hours.

Dr. Dwyer said that he had now treated one hundred and forty cases with the Hiss extract of leucocytes, most of them having had erysipelas, and the more he uses it in these erysipelas cases the more he is inclined to recommend it. The Germans are using it in cases of otitis media purulenta acuta. He has been treating his cases with five grains of urotropin every three hours until the secretions dried up.

Dr. Kenefick inquired whether the efficacy of the urotropin was confined to cases of streptococcus infection.

Dr. Dwyer replied that it was applicable to all cases of purulent meningitis.

Labyrinthine Disease Following Administration of Salvarsan.

DR. A. B. DUEL presented a patient, a young man 27 years of age. He had been in the United States Navy up to last April. Early in January he contracted a well defined case of syphilis. When the secondary symptoms began he was treated by the naval surgeon with the usual mercurials; the rash which had come out on his chest began to fade, and his hair, which had been dropping out, ceased falling when he was discharged from the navy. He then went to Baltimore, where his brother persuaded him to go to a surgeon or physician, who gave him "606." About a month later the patient suddenly became deaf and had a very decided vertigo which compelled him to go to bed, where he remained for three weeks. He said that "the sidewalk was not big enough to hold him, and everything whirled around in a certain direction." He could hardly stay in bed, but finally got up, though he is still a little dizzy at times. During this attack the patient had also a very decided impairment of vision. He could not say to what extent it was now affected, as he had seen him only once at the polyclinic, and had not been able to have any definite observations made on the eyes. The patient believed that he had entirely recovered his vision.

The case was particularly interesting from the fact that the patient has profound deafness and all vestibular reaction from caloric and whirling tests is lost in the left ear.

The doctor then produced a noise apparatus with which he tested the patient's hearing, showing that his hearing was very good on the right side, while he was unable to hear any sounds on the left side.

Dr. Duel, in connection with the report of this case, read a brief résumé of a very important paper recently published in the *Journal of Laryngology and Rhinology*[1] on the frequency of neurorecurrence in syphilis.

In 14,000 cases there had been a history of neurorecurrence in 126; 118 of these had occurred in the primary or early secondary stages. The recurrence had taken place in the second to the eighth cranial nerves, in the following proportion:

(1) **Benario. The Frequency, Causation and Therapeutics of Neurorecurrence After Treatment by Salvarsan—A Statistical Inquiry. Münch. med. Wochens., No. 14, April 4, 1911.**

Auditory, 43 per cent; optic, 26 per cent; facial, 15 per cent; the balance divided among the other four.

Benario strongly favored the opinion of Ehrlich, that the recurrences were due to syphilis and not to a toxemia. The explanation was that an insufficient dose improperly administered had left isolated colonies of spirochetæ in the nerve sheaths which subsequently started up a neuritis.

DISCUSSION.

Dr. Simpson said that while in Germany this summer, in conversation with a well known American ophthalmologist and aurist who had consulted Ehrlich on this very point, viz., the recurrence of certain symptoms after giving "606"—in line with the last extract read by Dr. Duel—Ehrlich expressed the opinion that undoubtedly in certain cases "606" does set free some additional syphilitic poison, causing these symptoms of remote infection to appear, but that if the "606" is subsequently repeated it will overcome this additional poison. That was his explanation of such cases as that reported by Dr. Duel.

Dr. F. C. Ard said that the night before he had heard a paper on salvarsan by Dr. Fox, who had studied a hundred cases very carefully, and who stated that he no longer had any fear of cranial nerve complications. In his opinion the complications that had been reported resulted from preparations that had preceded the use of salvarsan—preparations that had been used in the experimental stage. The cases which he reported had been very closely observed, the eye-grounds studied carefully in each case, and no complications had occurred, with the exception of three or four cases of iritis.

Dr. Kerrison said that a clinic case at the Manhattan Eye and Ear Hospital, which he had examined with Dr. Page about a year ago, illustrated one of the points brought out by Dr. Duel. This patient had contracted syphilis and seven months later became deaf in both ears and very dizzy. He had not received "606" treatment nor any injection, but only the usual mercuric treatment by mouth. He was absolutely deaf, both by air and bone conduction. He described vestibular symptoms as having occurred at the onset of the attack. These

symptoms, however, had largely worn off when he came under observation. He was still dizzy, however, and walked with considerable uncertainty. The caloric reaction was absolutely negative in both ears. He was revolved in a revolving chair ten times in each direction, with absolutely no nystagmus and no subjective disturbances, i. e., dizzines, ataxia, etc. In this case the symptoms were obviously due to the disease itself. Had he been given salvarsan and the symptoms appeared subsequently, they might have been attributed to the treatment rather than to the disease itself.

Dr. Berens said that he had just asked Dr. Harris whether he remembered some cases they had seen together before the days of the vestibular testing apparatus—cases of complete deafness in syphilitics. He recalled three, and there were possibly five or six more of these cases of complete deafness from syphilis long before salvarsan came out. All of them improved under iodid of potash, but not mercury. One case especially had taken an ounce of iodid of potash a day, and went up to three ounces a day, with a very marked improvement. One should not blame this deafness to a neurosis from arsenic.

Dr. Duel said that the advent of this case together with one other, and a rapid review of recent literature, had taught him that if we are to use the arsenic preparations in their treatment, salvarsan is much the safest one, and that if one dose does not succeed a second one should be given early, in order to prevent neurorecurrences. It was also very interesting to learn from Benario's report of so many cases that a majority of them recovered.

Dr. Guentzer inquired whether the dose had been given intravenously.

Dr. Duel replied that it was an intravenous injection.

Dr. Guentzer said that if it is true that an intravenous injection of salvarsan is eliminated in three or four days, it may be interesting in the diagnosis to know whether a patient had an intramuscular or an intravenous injection. Apparently the intramusclar injection allows the arsenic to pervade the system for a long time—some weeks or months; and it might be well to bear in mind whether it was really syphilis in itself or a toxemia from the arsenic, when considering a

diagnosis in these acute cases where an intramuscular injection was given.

Dr. T. J. Harris referred to a case which he has under care at present, which Dr. Strong knew about, and said that he wished to emphasize the importance of getting as much literature on this subject as possible—in regard to salvarsan in ear troubles. In this case the patient, who is forty years of age, has been suffering from progressive deafness. A diagnosis of otosclerosis was made. There was a straight history of syphilis. There are no symptoms whatever of syphilis now. A Wassermann test had been made by Dr. Strong, who reported it positive. The matter was placed before the patient and he accepted the possibility of having the condition improved by a salvarsan injection, which was given. Dr. Harris said that he was very much interested to know the effects of salvarsan in otosclerosis. Syphilis is recognized as one of its causes, but since he has heard of the possibility of injury to the ear from the use of "606" he is especially interested to know whether he is adding any additional risk in this respect. The patient has absolutely no symptoms of syphilis of the ear—simply slow progressing deafness, which has resisted treatment in the hands of very excellent men.

Dr. Libman said that it might be useful for the Section to know that the most complete discussion of this subject had recently been given by Delbanco and a collaborator in the *Muenchener medizinische Wochenschrift.*

Dr. Kenefick said that he recalled a number of cases of sudden deafness coming on late in syphilitic cases which had been reported before the otologic section and at the New York Otological Society, which yielded to heavy doses of iodid of potash and also of pilocarpin, but as he understood Dr. Duel's extracts, the cases reported were treated in the secondary stage.

Dr. Duel replied that practically all—118 out of 126—had been in the primary or early secondary stage, the very large proportion being in the early secondary.

Dr. Kenefick said that it would make a difference whether we were treating syphilis with salvarsan in deafness of the secondary stage or of the late tertiary stage.

Report of a Virulent and Rapid Infection of the Middle Ear and Mastoid Followed by an Evanescent Cellulitis of the Neck and Streptococcemia.*

By John Randolph Page, M. D.

New York.

DISCUSSION.

Dr. Libman said: The report of Dr. Page of the results obtained by Dr. Strong confirm to a great extent the observations that I have made for many years. I have been getting together all the data we have on this subject in order to present it for another association, and therefore at the present time I will simply discuss Dr. Page's case and give a few notes on some of our recent experience. It is very difficult to say whether or not Dr. Page's case also had a sinus thrombosis. As far as the temperature is concerned, it cannot be accepted or denied, for even afebrile sinus thrombosis has been described. However, if one does not wish to take for granted that a sinus thrombosis was present, it is possible that the streptococcemia developed as a result of cellulitis of the neck. In any case of cellulitis or of furuncle, a general infection may arise. It is fortunate that the evidence of the cellulitis appeared early, otherwise the sinus might have been unnecessarily attacked. It is most important in every case before operating on the sinus to exclude every other source of infection, unless symptoms of sinus thrombosis are very clear.

In the last year I have selected some cases which have been very difficult to interpret. In one case which I did not have the opportunity of seeing, the doctor considered that the patient had sinus thrombosis and asked for a blood culture. Streptococci were obtained, but in view of the clinical picture, the temperature not being very high, and the culture having been taken within twenty-four hours after operation, I advised taking another culture before operating upon the sinus. The second culture was negative, and the child recovered. The streptococcemia in this case may have been postoperative, or there may have been a sinus thrombosis.

In another case a physician suffered from purulent dis-

*See page 156.

charge from the nose following scarlet fever and bilateral otitis media. A blood culture was taken and a streptococcus found. The case was very difficult to interpret, for the man had had scarlet fever, and streptococcemia has been described as following scarlet fever with no marked focus of entry. There was also a possibility of the streptococcemia being due to the nasal infection, and a third possibility of its being due to otitis media. In that case I advised waiting for the report of the second culture. The second culture was again positive. It was agreed that it would be safer to explore the mastoid, for if the streptococcemia was of nasal origin, nothing could be done. An exploratory operation was done by a general surgeon. When the sinus was exposed the flow from the lower end was slow at first and later about normal. Meanwhile the pulse had risen to 182, and it was considered advisable to desist from further operation. The streptococcemia continued, and I was asked whether I would advise ligation of the jugular vein. As the surgeon had not in any other cases explored the jugular bulb, and as we were not sure that the infection came from the ear (there were only a few purulent cells in the mastoid), I advised giving the patient a chance to recover spontaneously if he had a sinus thrombosis, for it is well known that cases of sinus thrombosis have healed without operation. The patient recovered, and we do not know at the present time whether or not he had a thrombosis.

The third patient was operated upon for mastoiditis of the right side by Dr. Whiting. A few days later he developed a high temperature and marked swelling of the lymph nodes below the angle of the jaw on both sides. Because of the presence of these enlarged nodes, it was decided to have the nose and accessory sinuses examined. There was found to be present acute ethmoiditis and sphenoiditis of streptococcus origin. As it is posible that the streptococcemia came from this source, no further operation was done, and the patient recovered.

These cases show the necessity of looking carefully for the presence of primary foci.

In a fourth case, a patient was operated upon for mastoiditis, and four or five days later the temperature rose to over 105° and pneumococci were found in the blood. As we

had not had any cases of sinus thrombosis due to the pneumococcus, no further operation was done. The temperature lasted for several days. No cause was found, and the patient recovered.

A fifth interesting case is at present in the hospital. The patient had had otitis media for some time. He was operated upon for some nasal condition; the following night he had a chill and high temperature, and streptococci were found in the blood. It was thought likely that the infection was of nasal origin, but as he also had an otitis media, it was considered safer to explore the mastoid to see if the focus there was not being overlooked. The mastoid was found negative. The patient is still running temperatures and the eventual outcome is doubtful.

These cases, all coming within one year, have drawn our attention to the necessity of excluding disease of the nose and accessory sinuses before operating for sinus thrombosis upon patients who are suffering from otitic disease.

Dr. Seymour Oppenheimer said that the last case referred to by Dr. Libman showed the importance of excluding all other foci for the development of bacteremia. This patient had been admitted to the hospital the day after a nasal operation had been performed. The patient showed slight meningeal symptoms. In the course of the routine examination a blood culture was taken. For two weeks prior to the operation he had had an acute aural suppuration. The blood culture showed a bacteremia, and in view of the facts that there were no symptoms suggestive of a thrombosis of the sinuses adjoining the nose and that there had been an aural suppuration antedating the nasal condition, it was concluded that there was a possibility that there might be a focus remaining in the sigmoid sinus, although the tympanic condition had cleared up. Exploration of the sigmoid sinus was, therefore, decided upon, and performed rapidly. The wall of the vessel appearing normal, the wound was closed up. There has been no further development of symptoms suggestive of a thrombus in any of the venous channels in connection with the nose; and it was his impression that in these cases of infection from the nose, contrary to the route by which infection takes place in the ear, i. e., by the venous channels, infection may take place by the lymphatic channels.

DR. KENEFICK said that the point made by Dr. Oppenheimer was a very good one to bear in mind during the winter, and especially during the influenza season, when there are so many purulent conditions of the nose.

Paper: Streptococcus Infection and Immunity.*

BY L. W. STRONG, M. D.,

NEW YORK.

DISCUSSION.

DR. DWYER wished to congratulate Dr. Strong upon the production of such an interesting paper. It was too deep and contained too much interesting material to be discussed offhand. Certainly, clinically, the antistreptococcus sera have not proven of much value generally. There have been isolated successful cases reported, but, on the whole, the results have not been convincing. Theoretically, from what we know of the biologic activities of the streptococcus, it is hard to conceive that such antistreptococcic sera are to any high degree bactericidal or bacteriolytic. Certainly, Dr. Strong's observations on the streptococcus toxin are very interesting, and it is to be hoped that he continues his investigations along such lines. One point he wished to emphasize, as Dr. Strong had done, and that was to begin the use of any serum early and not wait until late in the course of the disease.

DR. LIBMAN said: I will touch on only a few points which Dr. Strong has brought up in the report of his investigations which are partly along the same lines that I also have been taking up. I agree with Dr. Strong that there has been an error in most of the bactericidal studies thus far done because optimum media were not used. A few years ago, in the course of the study of the bactericidal properties of the blood in a case of endocarditis, I found that one could get different results by the use of plasma and serum. Some years later Much of Hamburg showed that the effects of plasma and serum might be almost contrary, with certain organisms.

The point that Dr. Strong has made concerning the method of treating patients with infection is an important one. It

*See page 189.

is curious to see that patients with severe infections are allowed to go along for a number of days until they seem hopeless, and then everything is done at once. It is surprising to see how patients stand it. One must be careful in judging the results of treatment with serum and vaccine in all cases of infection. It is remarkable what most severe cases will do when nothing is done that may be of harm.

We have been quite interested in the question of transfusion in cases of severe infections. We are afraid to do transfusions in cases in which there are many organisms in the blood. There would be too much danger to the donor. According to our experiences, the transfusion does not do anything directly to the infection, but it strengthens the patient. Our present opinion is that if the patient does not have bacteria in the blood, it is advisable to do a transfusion in the hope of giving him a chance to make a better fight.

DR. STRONG said that he had only to add that the present streptococcus serum of the Board of Health is not standardized in any way; they are working on that now, but they make no effort to determine it by opsonic contents.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of November 10, 1911.

A Case of Sinus Thrombosis Without Subjective Ear Symptoms.

DR. EDWARD LEE MEIERHOF. The case is presented to show the necessity of examining the ears in cases of obscure diseases in adults. This patient, Rose R., twenty-one years of age, was under the care of her physician for four days, suffering from severe headaches and fever. As she had already been ill a week or more, and being too poor to afford a nurse, her physician had her admitted into the hospital with a tentative diagnosis of typhoid fever. An examination of the patient in the hospital, including the Widal test, was negative for typhoid. As no special cause for her condition could be found, I was asked to examine the ears, merely as a routine measure, although the patient did not give any evidence subjectively, she being perfectly lucid, of any ear involvement. An examination of the ear revealed a small granulation mass at the bottom of the canal of the left ear, but no signs of free pus. The swab used to wipe the granulation had a very foul odor. There was some tenderness over the mastoid on deep pressure, on a line corresponding to the roof of the external auditory canal; this, with a history of a chill and a rapid rise of temperature to 104 and 105 degrees every twelve hours, caused me to arrive at a diagnosis of pyemic sepsis due to an involvement of the lateral sinus of the left side. This diagnosis was confirmed by finding the dura bathed in pus from a sloughing thrombus and sinus. The removed jugular vein did not seem to be affected. This is the third case of the kind among adults that has come under my observation. Had this patient remained at home, I am sure she would have perished from sepsis from an unrecognized cause.

Case of Cerebral Abscess.

DR. DAVID G. YATES said that it was not his intention to report the case at length, but that he would show the specimen and give a brief outline of the clinical history.

The patient, a lad of sixteen, with a tuberculous family history, had had a running ear on the right side for two years, facial paralysis and slight vertigo for two months, earache and headache for a week. Two days previous to his admission to Dr. Kenefick's service at the New York Eye and Ear Infirmary, he had an attack of vertigo and vomiting. He had been under treatment at various institutions and with several physicians, until finally he came to Dr. Mulholland, who immediately referred him to me for operation. The principal symptoms noted on admission to the ward were pronounced facial paralysis and rotary nystagmus corresponding to the direction in which the eyes were turned, perhaps a little stronger to the affected side. Coordination was good. The patient was dizzy at times, but there was no nausea or vomiting. The mastoid was not tender.

On May 27th a radical (Schwartze-Stacke) operation was done, revealing a sclerotic mastoid, a small antrum lined with black necrotic membrane, granulations and carious bone about the mouth of the eustachian tube, and a few granulations in the region of the oval window, which were not disturbed. No dura was uncovered, and the facial nerve was not exposed. During the ten days following operation the patient seemed to be making a satisfactory recovery. The facial paralysis improved so that he could nearly close the eye, and the nystagmus was considerably less. At the end of this period of improvement, however, he suddenly began to vomit and to complain of intense vertigo, occipital and frontal headache and photophobia. It was found that dizziness was increased by lying on the left, or well, side. Careful examination now elicited the following additional particulars: Mentality was clear, but enunciation was delayed and somewhat indistinct. (This speech difficulty was peculiar; he seemed preoccupied by his dizziness and headache, and concentrated his thoughts with difficulty; he stumbled over the pronunciation of long words, but there was no real aphasia.) Reflexes were normal or slightly increased on both sides. A slight degree of muscular weakness and ataxia appeared on the affected side. Kernig's sign absent. There was a distinct tendency to fall to the affected side when attempting to walk; pronounced nystagmus and diplopia when looking to the right. Caloric irritability and hearing were present. He was not

able to retain food, and the bowels were obstinately constipated.

It will be noted that the symptoms which pointed strongly toward a cerebellar lesion as opposed to labyrinthine suppuration were the tendency to fall to the right and the nystagmus in the same direction. Had the lesion been purely labyrinthine, the reverse would have been the case. Nevertheless, we could not eliminate the labyrinth entirely, at least as a possible channel of infection, and it was determined to investigate this and explore the cerebellum at the same time. With this intention the wound was opened up on June 6th. Pus and granulations were found in the vestibule, but not in the semicircular canals. A flap was then cut to give access to the posterior cerebellar area, but the patient's condition made it seem wiser to postpone intracranial exploration. This was done four days later, the patient in the meantime having ceased vomiting and being able to retain food. The cerebellum was uncovered, about two inches back of the lateral sinus, through a bone opening three-fourths of an inch in diameter. Four attempts to locate pus by means of a narrow bladed knife and a grooved director proving unsuccessful, the anterior aspect of the cerebellum was exposed in front of the lateral sinus. No better success attended our efforts here.

The specimen shows the abscess to have been pretty deep, but it should have been reached from in front of the sinus. The latter, however, was situated rather far forward, and there was little room in which to work. The patient died three days later, and autopsy revealed an abscess involving the right cerebellar lobe, including the vermiform process, and extending quite to the median line. There are two cavities, one lined with an abscess membrane, the other not so lined.

The autopsy findings correspond closely with the clinical features of the case. There was probably an old quiescent abscess which gave no distinct symptoms until an acute process and our operative procedures stirred it into activity. I believe the facial paralysis was a middle ear affair and not due to the abscess.

DISCUSSION.

Dr. Dench said that he had seen the specimen at the New York Eye and Ear Infirmary, and that the case reminded him

of one which he had reported a few years ago, a case of tumor of the auditory nerve trunk complicated by cerebellar abscess.

In his own case he had missed opening the abscess cavity by only a short distance, and this had been the experience of Dr. Yates. If an abscess cavity is situated at the cerebellopontine angle—and this is the location in which most of these abscesses are situated—the purulent focus may be reached by incision in front of the lateral sinus, provided the lateral sinus is situated rather far backward. If the abscess cavity is entered in front of the sinus, it is very difficult to secure an opening large enough to afford proper drainage. In all cases where the abscess cavity cannot be reached in front of the sinus, it is wiser, instead of attempting to uncover the entire cerebellar hemisphere by enlarging the bony opening backward over the lateral sinus toward the median line, to disregard the original mastoid incision, and to expose the cerebellum by turning down a large flap, exactly as one would do if he wished to expose a tumor at the cerebellopontine angle. It is not wise to perform an osteoplastic flap in exploring for a cerebellar abscess, because the soft structures over the cerebellum are sufficiently thick to provide an adequate covering for the brain, even if all the bone is removed. Exposing the cerebellum in this way, the cerebellopontine angle can be easily exposed by lifting the cerebellar hemisphere upward, outward toward the median line, thus exposing perfectly the cerebellopontine angle, which is the favorite site for these abscesses, and also exposing the internal auditory meatus, which is the most frequent avenue of infection in cases of cerebellar abscess. The advantages of this procedure are that it affords a free exposure of the area most frequently involved, and prevents possible injury to the lateral sinus which, if it occurs, either greatly delays the completion of the operation, or may render its completion absolutely impossible.

Dr. Dench said that edema of the upper eyelid was not characteristic of pus formation in diseases of this character. An edema of the eyelid frequently follows a supraauricular incision, and is not infrequently found on the second or third day after the ordinary radical operation, provided the incision has been carried well forward over the ear.

Dr. Phillips said the case to which he had referred had edema of the eye before operation.

DR. DENCH said that in his opinion whether edema was caused by a pathologic process, such as a deep temporal abscess, or whether it was caused by stitches inserted for wound closure, made no difference. The point that he wished to emphasize was that edema of the upper lid might follow involvement of the superficial structures in this region from any cause whatever.

DR. PERCY FRIDENBERG said that the question of symptomatology was obscure at best, and Dr. Yates was to be congratulated on the careful analysis of his case. Anything that will aid in the diagnosis of cerebellar abscess should be welcomed, and as the number of correctly diagnosed cases increased, the interpretation of clinical signs and symptoms would become more and more accurate. Two tests had been worked out by Bárány within the last year or two, and according to the statements of this investigator, were valuable aids in the diagnosis of cerebellar lesions and their differentiation from functional labyrinthine affections. The first was the influence of head position on the direction of falling, either with spontaneous or rotational vertigo. If there was a tendency to fall to the right, turning the head to the left changed this into a tendency to fall forward; turning it to the right caused a tendency to fall backward. If there was a tendency to fall to the left, this was changed, similarly but in the opposite sense, by head turning. This reaction is quite characteristic, and its presence or absence is of pathognomonic significance in the differential diagnosis of cerebellar and labyrinthine vertigo, or, at least in the determination of the presence or absence of a destructive lesion, such as abscess, in the cerebellum. The second test was known as the pointing deviation test. It was quite similar to that used by ophthalmologists in testing the innervation of the muscles in a case of ocular palsy. When an attempt is made to move the eye toward the side of the paralyzed muscles, an excess of innervation is required. This is interpreted by judgment to indicate a position far off to the side of the object fixed, and so these patients, when told to point at the object they are fixing in abduction, "over point" with the finger, i. e., they deviate to one side. Bárány found that the control for all such motions, whether carried out by the upper or by the lower extremity, was located in the cerebellum. After rotation and the induc-

tion of rotational vertigo and nystagmus there is regularly a characteristic pointing deviation, of finger and hand or of toes and feet, to one side. With a destructive lesion of the cerebellum on one side this deviation is lost and rotation has no effect on pointing with the hand corresponding to the cerebellar lesion, while deviation is characteristically present when the other hand is used for pointing after opposite rotation. Bárány claims to have been able to differentiate between the two sides of the cerebellum in a number of cases of Horsley's, and read an extensive paper on these tests before the British Medical Society last summer. The speaker would like to know whether Dr. Yates had applied either of these tests, and whether other members of the Section had come to any conclusions as to their practical value.

Dr. Duel said that the tests of Bárány just alluded to by Dr. Fridenberg would have been very interesting in this case. The symptoms as they had been narrated by Dr. Yates pointed very definitely to a cerebellar abscess, but would hardly seem to warrant the suspicion that the cerebellar infection had taken place through the labyrinth. Had the patient shown a nystagmus the quick movement of which had previously been away from the diseased ear, subsequently developing a nystagmus with the quick movement directed toward the diseased side, such a diagnosis would have been probable. In such a case a caloric test would probably have shown that the labyrinth was not functionating on that side. But with a nystagmus the quick movement of which was directed toward the diseased side, no history of a previous nystagmus in the other direction, and giving a normal reaction to the caloric test, one would not be justified, in his opinion, in doing a labyrinthine operation as a preliminary to the exploration of the cerebellum. The shock of the labyrinthine operation added to the cerebellar exploration would be a serious and unnecessary risk to assume in such a case.

Dr. Perkins said that in one hundred cases of cerebellar abscess reported from 1898 to 1906, analyzed with reference to etiology, etc., one-third were traced with reasonable certainty to infection through the labyrinth; in another third, by way of the sinus; in the remaining third, the route of infection was not stated or unknown. In many of these cases facial paralysis was found, so that it seems reasonable to suppose

that in a certain number of cases infection occurs through the fallopian canal.

DR. EAGLETON said that he thoroughly agreed with what had been said about the thorough exposure of the cerebellum, such as is necessary for the removal of tumors of the pontine cerebellar angle. Otologists are losing many cases of cerebellar abscess which they will learn to save as soon as such are approached as a distinct entity and not accidentally diagnosed at the time of the operation or when the patient is in extremis. If an early positive diagnosis is made of the cerebellar abscess, and it can now be made with a great deal of certainty in many of the cases, and if it is approached through a large opening, going behind and making a large dural flap and thoroughly draining the abscess from behind the sinus as well as in front of it, instead of making a small inefficient opening in front of the sinus as they are now doing, and thus approaching it at great depth, we will save a great many cases which otherwise would be lost. In a recent case he had operated upon—a case of temporosphenoidal abscess—the ear discharging, he went above and made a large opening with a dural flap, packed off the subdural space as one would the peritoneum, explored and found pus, used the encephaloscope and through it carefully drained every drop of pus, so that a perfectly clean dry cavity remained, put in a gauze drain and left it for twelve days, when the last wick was removed and never replaced, the child made an uneventful recovery. In another case of cerebellar abscess, after having found the abscess by exploration from in front of the sinus, the sinus being so far forward that it was impossible to thoroughly drain this way, an effort was then made to explore it from behind, during which procedure the abscess cavity was lost, which will always result if we have not room enough to thoroughly inspect at the time of primary puncture and while the pus is still flowing, and later try to further expose after locating the abscess. In such cases the gauze drain which is put in is never properly applied into the abscess cavity. It is simply passed into the brain as in this case, and afterwards it is found that the cavity has refilled. The vast majority of such patients die, as in this case.

As to the diagnosis of cerebellar abscess in relation to nystagmus, we must remember that we do not have nystagmus

in cerebellar abscess unless the vestibular tract is irritated, which only occurs either by an abscess situated deep in towards the median line or near the auditory meatus. If it is external, in the lateral lobe—in the latent area, as it is called —we do not have nystagmus. In the case to which he referred the patient, a girl, at the time of operation had no nystagmus, but three days afterwards she had a marked nystagmus, due to the extension of the abscess cavity deep in towards the median line, as was found at the postmortem examination.

Dr. Johnson said that it would be very nice when we get to the point of being able to determine satisfactorily and at once the question of the diagnostic significance of the symptomatology of these conditions, but so far as his experience goes it is very difficult to decide, and many men are misled by different symptoms indicating various conditions during the progress of the disease. The doctor had indicated in his report the doubt and the uncertainty that one feels when some new difficulty arises. As to the matter of the response to tests, very frequently you do not get satisfactory responses, although cerebral abscess is subsequently known to have been present. In one case which Dr. Eagleton had seen with him this summer, and which terminated disastrously, the patient, a young man, had been operated upon for mastoid disease two or three years previously. He came complaining of silght headache. He had had no discharge from his ears for two years, until about ten or twelve days before coming for treatment, and had then a slight discharge from high up in the middle ear, which was quite odoriferous. He complained of pain in the back of his head, and said that he had been examined for life insurance and the examiner had said that it would hardly be right to recommend him for insurance, as he might at any time develop a brain abscess. Immediately after that he began to feel very uncomfortable, but Dr. Johnson thought at first it was simply induced by the statement of the individual who had examined him for life insurance, the trouble being of an hysterical character. The symptoms, however, continued to increase and the headaches were more constant. At the same time he had severe inflammatory nasal trouble with pain and tenderness over the frontal sinuses, fever of a remittent type, and his symptoms could all have

been caused by a frontal sinusitis. He went to bed and stayed there for two or three days, and felt much better, his temperature became normal, and he went out and down town and did more or less business. After the second day, however, he had a return of the headaches, dizziness, and slight nystagmus, and then two days later first complained of the peculiar movement of the picture molding on the wall—probaby it was the movement of his eyes in the nystagmus which was away from the affected ear and of increasing intensity. He was sent to the hospital, and the second day following Dr. Eagleton saw him in consultation. The spinal fluid was examined and found to be normal; it was concluded, however, that he had a cerebellar abscess. At that time his condition was very bad, his nystagmus was more marked, but he did not respond to the caloric or other tests which would indicate that it came through the labyrinth, although it seems probable that it did come from extension to and through the labyrinth. On commencing the operation, while taking an anesthetic, the patient ceased to breathe, the operation was stopped until he was brought around. It was decided to rapidly open up the abscess over the cerebellar point, the point suggested by Dr. Dench. Immediately after the cerebral cavity was entered there was a spontaneous gush of pus, and a tremendous cerebellar abscess was evacuated. The shock of the hammer was so objectionable that a trephine should be used to make the opening in such cases. Although up to four days before the operation the patient had had very few symptoms. Even on the morning of the operation, although he had been at times a little slow mentally, he had spoken clearly to a nurse whom he saw for the first time since his mastoid operation, and his mental perception seemed quite clear, and he and his family thought he was much better. He died immediately on the sudden exit of the abscess contents. It was a very interesting case, and very obscure as to the conditions indicated by the symptomatology. No autopsy was allowed, but an extended examination showed an abscess cavity about four inches deep extending across the cerebellum and apparently walled off.

Dr. Yates said that he had learned a good many lessons from this case, one of which is that one should make a thorough exposure of the cerebellum when exploration is undertaken.

Replying to Dr. Fridenberg: he had not made the tests referred to.

Dr. Duel's criticism was just, but the course pursued was not followed without consultation and careful consideration of the case. As a matter of fact, he had not been able to reconcile the symptoms, and had not detailed them all, as it would have required too much time.

A New Noise Apparatus for the Exclusion of Hearing.

Dr. Francis W. White said that the apparatus which he presented looked somewhat formidable, but in reality the instrument itself was not.

It was simply a cut off or stopcock made of hard rubber, to which had been joined a short tube about an inch and a half long of the same material, having a small oval opening upon the upper aspect about the middle. The opening is ¼x⅛ inch.

The rest of the paraphernalia is a Paquelin cautery bag and stethoscope; the former being used to give the required air pressure, the latter to augment the sound produced by the air passing through the cut off. By means of the cut off the volume of sound can be increased or diminished as required by an individual case. Instead of the stethoscope, a diagnostic tube may be used. To demonstrate the instrument, he used a stethoscope, however, but unfortunately a case of unilateral deafness, which should have been here, has not arrived. Naturally, a case of this kind is the better for demonstration.

The cautery bag is attached to the short end of the cut off and the air compressed. The stethoscope is attached to the long end of the cut off, and the ear tips are accurately fitted into the patient's ears. The cut off is then gradually opened until the patient fails to hear the examiner speaking, even when the voice is raised so that actual shouting is resorted to. Manifestly, if the patient hears nothing under these circumstances, he cannot possibly hear in the ear we wish to eliminate, provided the cut off is left exactly as when both ears were examined. To examine one ear, simply remove the tip from it, preventing the escape of sound from the corresponding tube of the stethoscope by means of a small hemostat or similar instrument, and proceed with the examination. For the other ear reverse the maneuver.

This particular arrangement is intended for use outside of one's office, as in the office the cautery bag can be dispensed with and pressure by means of air pressure tank used, simply attaching same by means of a small piece of rubber tubing and allowing the patient to quietly sit and hold the instrument himself.

Dr. White said that the advantages of the apparatus were:

1st. It effectually cuts off hearing.

2nd. It can be regulated to suit each case.

3rd. Bone conduction can practically be termed nil, as it is so slight.

4th. No disagreeable sensations after its use.

5th. Simplicity, easy transportation, and small cost.

Everyone who has used the Bárány apparatus knows that patients complain of the severe noise during examination and a sense of deafness after examination, also of a ringing in the ear examined. The bone conduction is also excessive and lessens the distance at which a patient should hear with the ear under test.

In a case of acute labyrinthitis these things are very important, as the patient is very ill and naturally resents any disagreeable procedure, but we certainly desire to know whether he hears much, little, or not at all, while we are trying to make a diagnosis of this condition.

The apparatus operated by means of water naturally suggests many obstacles.

The method of covering the ear with the open hand and percussing with the fingers of the other can be used in a very limited number of cases and only in a superficial examination.

Replying to a question from Dr. Dench as to what produced the sound, Dr. White said that it was produced by the rush of air through the cut off which threw the air into swirls, thus producing a sound, the latter being transmitted and augmented by the tubes leading from the cut off, while the air escaped from the oval opening, as shown in the instrument.

Answering Dr. Phillips, who asked if the metal portion of instrument augments the sound, Dr. White said that he thought possibly it did, but by using a heavy tubing the stethoscope can be eliminated, although the average diagnostic tube did not appear to be made of heavy enough rubber.

In the absence of a patient for demonstrating the instrument, many of the members tried it personally.

DR. PHILLIPS said that from the sound which he heard at a distance there did not seem to be enough air pressure.

DR. DENCH said that he thought the objection in regard to the bone conduction of the Bárány apparatus was correct, and he had never believed in it thoroughly for that reason. One gets a good deal of bone conduction.

DR. WHITE said that not only was that true (in regard to bone conduction of the Bárány apparatus), but that the patients complain of buzzing and ringing in the ears afterwards. With this apparatus one can put a great deal of pressure in and there is no disagreeable effect.

DR. HELLER said that not having a Bárány apparatus in his office, and wishing to make a test of hearing, he had used the ordinary Wappler pneumomassage apparatus, which most otologists have, employing the to-and-fro motion and speeding it at a high speed. He found that this blocked out the one ear very successfully. He would not recommend this as a routine procedure, but as he had used it it answered very nicely. He had not screwed up the valve very tight, but let a little air in, setting the pump at almost full speed, and it worked as well as one could expect the Bárány apparatus to work.

DR. DENCH tested the apparatus personally, and said that it was the best thing of the kind that he had seen.

DR. PHILLIPS said that the instrument which he uses is known as the Neumann apparatus, and works on the principle of the telephone receiver. This instrument absolutely shuts out the sound, but there is a great deal of buzzing which is unpleasant to the patient. If this apparatus does cut out the sound completely it is very desirable.

DR. EAGLETON said that he wished to take exception to Dr. Phillips' statement. Neumann's apparatus generally completely cuts off the sound from one side. It does so in the majority of cases, but not in all. He had borrowed Dr. Phillips' instrument and was so well pleased with it that he wrote to the manufacturers in Vienna and had them send him two, and while it worked well in the majority of cases, it was not always to be depended on to thoroughly cut off all the sounds from one ear.

DR. DENCH said that he did not think one could cut off all the sound transmitted to the healthy ear, for you do get some bone conduction. He did not know of any way in which one

can shut out all bone conduction with a perfect ear on one side and a bad ear on the other; he did not know of any instrument that would cut it all off. This one does it better than any, and, as Dr. White had said, it was exceedingly simple.

Dr. Johnson said that he thought it would be well if the ends of the stethoscope were placed well in the ear canals. When the tests were made at Atlantic City by Dr. Pierce, he insisted that the tips of the tuning forks which he used for this purpose should be pushed well into the ear. It is very uncomfortable to have the ends of a tuning fork jammed into your ear. With the apparatus shown tonight hearing through the nasal passages, or the bone, or anything else seemed to be lost; he could see the doctor's lips move, but could not tell whether or not he was making a sound. It seems to be a question of making these experiments very carefully. It is very difficult to get an apparatus that we can be absolutely certain of excluding all sound, for the perception of sound through the bone or nasal cavity might vary in different individuals and under different conditions. The doctor's apparatus has the advantages of cheapness, comfort in use, simplicity; and seems to exclude sounds of ordinary intensity.

Dr. Page said that Dr. White had spoken to him of an interesting point to which he had not referred tonight, namely, the effect of the apparatus in cases of perforation of the drum.

Dr. White replied that when first testing the apparatus he had experienced difficulty in excluding the hearing when a perforation of the drum was present, except when using an air-pressure apparatus common in the office of an otologist. At present, however, by using material heavier than the ordinary diagnostic tube, sound can be completely excluded with the pressure supplied by the cautery bag. He also called attention to another use for noise apparatus. In a case of malingering—one who claims to have become deaf after an accident, for instance. The patient is told to read from a book or paper. After having read a paragraph the noise apparatus is used and the patient is allowed to read again, when a sudden increase in the loudness of his voice is noticed. This is due to the patient not being able to hear the voice in its normal tone. If the patient is really deaf there is no change in the voice.

Deep Temporal Abscess.*

By Alfred Braun, M. D.,

New York.

DISCUSSION.

Dr. Phillips said that he had been very much interested in the pathology of this affection which had been outlined by Dr. Braun, but thought the doctor had not sufficiently emphasized one or two clinical symptoms—possibly because he had found that the location of the pus was so definite in the cases which he had seen, and the swelling so clearly defined. Perhaps he had not seen other cases that present other symptoms. He recalled several cases in which the swelling was chiefly confined to the areas of the muscle, but they certainly had edema, especially of the eyelids, on the affected side. He has seen this in several cases, one of which proved to be a temporal abscess. Another case developed a brain abscess, a temporosphenoidal abscess, showing that the pus went into it.

Dr. Dench said that the Section was indebted to Dr. Braun for giving a clear picture of cases which all have seen but have not described so clearly. He himself had not seen more than half a dozen cases of this kind since he has been practicing otology, and had never thought of describing them as a distinct class. Dr. Braun's paper was of great value in describing them as a distinct class.

Dr. Chambers asked if it was always necessary to carry the incision so far forward as Dr. Braun had done. He has had several cases where he has not carried the incision so far and had curetted out the diseased tissues with good results, in a place where one does not like to make so long an incision.

Dr. Hays, in connection with the cases cited by Dr. Braun, reported a case that he saw at the Infirmary. The patient had been operated upon for mastoiditis; the wound had healed nicely, but two months later she came back complaining of severe pain over the temporal region. There was considerable swelling in the region of the zygomatic process, which was tender on pressure. A diagnosis of syphilitic periostitis was made, the lesion clearing up on antisyphilitic treatment.

Dr. Fridenberg called attention to the fact that collateral

*See page 170.

edema alone causes a swelling extending forward along the zygoma as far as the orbital margin and even involving the soft tissues of the lids and eyesocket. Some exophthalmus and limitation of motility outward of the globe might also result. This condition was not infrequently seen with marked involvement of the zygoma cells, when there was no question whatever of a subtemporal abscess. Of course, in the cases he referred to, the swelling was not strictly limited to the temporalis muscle, so that the shape of the swelling might vary. It was mainly due to interference with return circulation, and accordingly depended somewhat on the vascular local supply and on peculiarities of the fasciæ in this neighborhood.

Dr. Braun, in closing, said that the edema of the eyelids, which may occur in these cases, is due to secondary circulatory changes, as Dr. Fridenberg stated. There is an obstruction to the return venous flow from the eyelids. But the shape of the swelling at the side of the head is characteristic. The swelling must take the shape of the temporal muscle, on account of the attachment of the temporal fascia to the bone.

Replying to Dr. Chambers' query, as to whether it was necessary to carry the incision so far forward, he said that it was absolutely necessary, in order to reach the temporal fossa and remove all the diseased bone in the zygomatic arch. The root of the zygoma can be reached through the ordinary mastoid incision, but not the temporal fossa. As the anterior part of the incision is sutured, and there is usually no difficulty in getting primary union, the scar, being entirely covered by hair, is scarcely noticeable.

ABSTRACTS FROM CURRENT LITERATURE.

I.—EAR.

The Etiologic Factors of Otitis Media Purulenta Chronica.

S. MacCuen Smith (*New York Medical Journal,* October 28, 1911) calls attention to the fact that there may be present at all times in the middle ear certain microorganisms, but that an exciting cause from without is required to cause them to do harm. The organ of hearing is almost wholly dependent upon the health of the general organism for the preservation of its normal functional activity. He believes that the greatest importance should be attached to perverted ventilation of the tympanic cavity as a cause of early discharge, and on that account lays greatest stress on proper nasal breathing, regarding any obstruction here as an important causative factor in chronic otorrhea and a standing obstacle in the work of repair.

Harris.

Acute Otitis Media—How Best to Treat it so as to Prevent Complications.

George L. Richards (*New York Medical Journal,* October 28, 1911), in a paper addressed to the general practitioner, gives many practical suggestions regarding the treatment of this common disorder. After calling attention to the importance of every physician being able to examine the ear drum, and the all-weighty role that the nasopharynx plays in producing middle ear diseases, he recommends for the first stage of otitis media the use locally of an aural bougie composed of carbolic acid, fluid extract of opium, cocain, atropin sulphate, water, gelatin, glycerin. Internally he employs granules of aconitin, 1/134th of a grain, combined with small doses of morphin, according to the alkalindal formula. He recommends early paracentesis where bulging has taken place, and in place of irrigation the employment of gauze wicks.

Harris.

Relation of Salvarsan to the Ear.

SIMON SEEGMAN (*New York Medical Journal,* December 16, 1911). The possibility of serious effects on the ear from the use of salvarsan seems to have impressed itself much more upon the European otologists than upon those in this country. At a recent meeting of the Otological Society in Vienna, ten cases were reported of ear complications occurring following the use of salvarsan, in the course of a few months. As a means of judging whether this was a mere coincidence or due to the use of the drug, the author refers to an investigation conducted a few years ago by Alexander, to determine the frequency of involvement of the ear in cases of syphilis.

As a result of six years of study of all the cases treated at the General Hospital and in the Polyclinic in Vienna, Alexander was able to find but six cases where the ear had been involved. From these the author concludes that the cases of ear affection following the injection of salvarsan were of necessity caused directly by the salvarsan. In addition, he reports the case which came recently under his own observation.

The patient was a seamstress, 30 years of age, who acquired syphilis in March, 1910. She presented herself between March and June for treatment, and received intramuscular injections of mercury. The latter part of September trouble began in the right ear, subjective noises, slight dizziness, increased with movement of the head or change of posture from reclining to standing. Upon examination both branches of the eighth nerve, the cochlear and the vestibular, were found to be impaired, eight months after infection. Left ear was normal. The patient took a course of mercury treatment under Dr. Zumbush. November 4th she was discharged, absolutely free from any ear symptoms, and hearing equally well with either ear, and no ear affection. Later in the same month the patient returned with the complaint that the right ear had remained well, but there was a diminution of hearing in the left, with noises. Examination showed the right ear to be normal, while the left showed the same symptoms previously shown in the right. She received an injection of salvarsan by Dr. Noble, with slight improvement in the hearing. Ten days after the injection patient again appeared, complaining of severe, lancinating pain in the left ear, radiating to back

of the neck. Hearing poor, noise increased intensely, two or three attacks daily of vertigo followed by profuse vomiting, increasing in severity upon changing posture from reclining to standing. When lying down the patient was free from vertigo. Upon examination the right ear was normal and the left had completely lost its function, there was loss of equilibrium; the semicircular canals did not respond to caloric or mechanical stimuli. Two months later partial facial paralysis on that side developed, positive to faradic current.

In view of this he feels that in all cases of syphilis searching inquiry should be made into the past and present condition of the ears, and, if possible, both the cochlear and vestibular branches should be tested for any impairment or loss of function. In any ear affection, whether due to syphilis or to any other condition, where the nerve is involved, salvarsan is contraindicted until further demonstration. *Harris.*

Woodeny Phlegma of the Sternomastoid Region.

M. P. Moure (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* July 15, 1911) reports a case occurring recently under his care. The patient was a woman of 23, who eight days before had been seized with a severe pain in the left mastoid region. The temperature was 38° C. The following day palpation showed the presence of four small glands upon the mastoid. They were mobile, but extremely painful. The next day the superficial glands over the external jugular became enlarged and painful, and soon a general adenitis of the neck occurred. Ten days later there was a swelling in the left sternomastoid region, which extended upward to the base of the mastoid, and downward to the middle portion of the neck along the sternomastoid, giving the appearance of a Bezold mastoiditis. The absence of all ear symptoms excluded this.

The local temperature was elevated, and pain was pronounced over the entire region. The inflammatory area faded insensibly at its periphery, without clear limitations, and included the entire region in an immovable mass of woodeny hardness. Under general anesthesia the swelling was freely incised. The sensation to the introduction of the knife was one of cutting through lard. There was free bleeding. The mastoid was found entirely normal. The sternomastoid mus-

cle was embraced in this inflammatory process. At almost the center of the incision, which did not give rise to any pus, a yellowish white mass the size of a small hazelnut was discovered. The culture in bouillon gave a streptococcus infection. The patient made an uneventful recovery.

The case is fairly characteristic of most of the cases reported of this rare affection. The duration was relatively short, four, five and even six months having been reported in some of the other cases. The absence of pus is peculiar to the disease, which is wont to occur only late. The careful laboratory investigations allowed the exclusion of actinomycosis. The streptococcus recovered was of feeble virulence. Emphasis is laid on the importance of early intervention after the diagnosis is made, in order to cut short the progress of the disease. *Harris.*

An Unusual Form of Mastoid Infection in Chronic Otorrhea in Children. (Mastoiditis Nigra.)

BRINDEL (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* September 30, 1911) calls attention to the fact that macroscopically there are various forms of mastoiditis, such as purulent mastoiditis, fungous mastoiditis, and osteomyelitic mastoiditis. In addition to these he has met in sixteen cases out of twelve hundred operations a form which he calls "Mastoiditis Nigra," and of which he gives the following description:

First, that mastoiditis nigra is met with in chronic infections of the mastoid, and is characterized by diffusing cellulitis of a blackish coloration.

Second, it is peculiar to infancy and is almost exclusively encountered in old otorrheas, especially fetid.

Third, the blackish coloration of the alveolar cells and of their contents does not exclude other lesions, which are wont to be met with in such cases, such as granulations, cholesteatoma, etc.

Fourth, mastoiditis nigra is a diffused cellulitis of the mastoid process, requiring almost always a complete removal.

Fifth, the postoperative healing in these cases does not differ from that in other cases.

Sixth, the pathogenesis of mastoiditis nigra is still undiscovered. *Harris.*

The Conservative Treatment of Chronic Suppuration of the Middle Ear, With or Without Involvement of the Labyrinthine Wall.

TRETROP (*Revue Hebdomadaire de Laryngologie,* etc., December 2, 1911) is a believer in the possibility of curing many cases of purulent middle ear diseases without operative intervention. He makes use of the latter whenever it is called for. He does not state under what conditions he is wont to interfere surgically, but states that it only represents 5 per cent of the cases treated by him. He places much importance on the use in the middle ear of the various medicaments containing oxygen, among these hydral, a concentrated solution of 100 per cent of oxygen, or pergenol, a tablet which when dissolved in water gives a solution of borated oxygen, and the peroxid of zinc, which gives rise both to oxygen and to the cicatrizing powers of the oxid of zinc. He claims for the oxygen a specific power in curing of suppuration by the prevention of anerobic microbes, and by the formation of increased phagocytosis. It is interesting to remark this enthusiasm in the use of oxygen, when it has been virtually abandoned in this country. He also employs with advantage antiseptic solutions, especially the glycerin solution of bichlorid of mercury, as well as a weak solution of formalin. Iodinated glycerin he employs frequently with good results.

Harris.

The Bacteriology of Sinus Thrombosis.

HERZOG, Munich (*Münch. med. Wochenschr.,* No. 50, 1911). The groundwork in this subject was originally worked out by Brieger and Leutert. Leutert's method of determining the presence of a sinus thrombosis consisted in the aspiration of 1 cc. of blood from the sinus and the same amount from a peripheral vein. If the former showed colonies on agar plates, while the latter remained sterile or showed decidedly fewer colonies, the diagnosis of sinus thrombosis was made. The sterility of the blood taken from the sinus did not, however, necessarily exclude a thrombus.

The fact that the peripheral blood contained so few bacteria when the thrombus was continually emptying them into the blood current was explained by the bacteriolytic power of the blood. Herzog, as a result of a carefully observed case, takes exception to these observations and draws the following conclusions:

Usually speaking, the wall of a sinus where infection is present is always infiltrated with bacteria. When the needle is passed through, and the blood aspirated into it, the small fragment of the wall which remains on the needle is at the same time drawn into the syringe and becomes mixed with the blood, thus giving it its rich content in bacteria. Thus when there is no real thrombus, as in his case, but a perisinus abscess with granulations on the wall, a false result could easily be reached.

In making such an investigation he proposes the following regulations:

Agar Plate No. 1.—1 cc. of blood punctured through the most diseased portion of the sinus wall.

Agar Plate No. 2.—The needle remains in place and 1 cc. of blood is again withdrawn.

Agar Plate No. 3.—Puncture as near as possible to the first position. Careful withdrawal of the needle without aspiration. Sterile salt solution is now drawn into the syringe and the contents put on the plate.

Agar Plate No. 4.—Sterile salt solution is again drawn up through the same needle and put on the plate.

Agar Plate No. 5.—Puncture of the median vein.

No. 1 contains the bacteriologic products of the wall plus the blood of the sinus.

No. 2 contains the bacteriologic products of the blood itself.

No. 3 contains the bacteriologic products of the wall of the sinus alone.

No. 4 should remain sterile.

No. 5 gives the contents of the peripheral blood.

A comparison of the various cultures would lead to a proper interpretation of the results. *Horn.*

Results With Treatment of Thirty Cases of Otitis Media by Vaccine Therapy.

C. L. McDONALD, Cleveland (*Journal American Medical Association*, June 3, 1911). In subacute cases, when the staphylococcus albus and the pneumococcus were the causative organisms, the results were all good, autogenous vaccines being used in every case.

In chronic cases the results were not as good, nine out of

seventeen cases showing no improvement whatever. Three recovered completely. It is to be used in chronic cases only when other measures have failed. The chronic cases sometimes stop discharging after one or two inoculations, the discharge appearing again at a later date, when inoculations will again cause cessation for a time. *Richards.*

Transillumination of the Mastoid Process as an Aid to Diagnosis.

EMIL AMBERG, Detroit (*Journal American Medical Association*, June 3, 1911), reports a case of acute mastoid disease in which transillumination by the method of Dintenfass was performed. This consists in the introducing into the external auditory canal of an electric lamp two cm. long by one and one-half cm. wide until only the cable winding is visible. This gives a picture somewhat similar to transillumination in other cavities, and by comparison with the sound side is a valuable aid to diagnosis, the light passing with difficulty through the inflamed or purulent mastoid, and easily through the sound mastoid. *Richards.*

Concerning the Etiology of Otitic Meningitis.

SHINI-IZI ZABA (*Archiv. für Ohrenheilkunde*, Vol. LXXXVII, p. 1-16) says that the usual belief concerning the origin of otitic meningitis is that in the large majority of cases it results from a suppuration of the labyrinth, which gains access to the meninges either through the internal auditory meatus or through the aqueduct. He reports three cases of otitic meningitis. The infection of the first case followed the above path; the other two, however, occurred in an entirely different manner, as the infection of the meninges developed without inflammation of the labyrinth. In one of these cases the cerebellar wall of the antrum was necrotic, and at the operation the cerebrospinal fluid ran out of the wound at a place where the inflammatory process had opened up the pia mater. In the other case the processes of the meningeal infection were somewhat more complicated. In this case the process was not an acute one, but a chronic suppuration of the middle ear with an extensive cholesteatomatous formation. This cholesteatoma, without touching the membranous labyrinth, reached to the internal auditory canal and

had formed there a large cystic tumor. From this deposit the infection easily spread to the meninges. The author calls our attention to the fact that these cases illustrate that the path of infection in cases of otitic meningitis may be extremely variable. *Wood.*

Atticoantrotomy.

SEJII KASHIWARBARA (*Archiv. für Ohrenheilkunde,* Vol. LXXXVII, p. 20-36) describes the operation of atticoantrotomy. After previous removal of the drum membrane and the malleus and incus he makes three parallel cuts through the membranous canal. The first one on the posterior wall at the height of the floor of the aditus ad antrum, as ascertained by the probe; the second on the middle of the superior wall; and the third at the junction of the superior and anterior walls. The two skin flaps thus made are raised by a bent raspitory and drawn outward and held in place by the introduction of a speculum. After the bleeding has been controlled by cocain and adrenalin, a small chisel is driven through the bone at the position of the cutaneous incision, care being taken that it does not break into the facial canal. Then, under careful guidance with the eye, small pieces of bone are removed in an upward and anterior direction until the cavity of the attic is laid wide open. After careful probing the floor of the antrum is located and the posterior wall removed so as to open it up. The operator, however, must be very careful not to go too far downward for fear of wounding the facial nerve. The antrum, however, should be sufficiently opened so that its cavity can be easily seen. The operation can then be extended, if desired, into the mastoid cells by continuing the removal of the posterior canal wall outward and backward.

Kashiwarbara says that the indications for simple removal of the drum and extraction of the malleus are: first, chronic suppuration of the middle ear with caries of the ossicles and cholesteatoma in the tympanic cavity; second, ankylosis of the malleoincudal joint; third, closure of the eustachian tube. Indications for the removal of both malleus and incus are: first, in light cases of middle ear suppuration in which the mucous membrane of the drum cavity is alone involved; second, carionecrosis of the ossicles; third, ankylosis of the malleoincudal joint with scarring of the mucous membrane

of the drum cavity. Indications for the removal of the stapes and closure of the oval window with an artificial membrane are: first, favorable cases of otosclerosis, and second, cicatricial closure of the oval window. Atticoantrotomy is indicated first in chronic suppuration of the middle ear where conservative treatment has had no results and where suppuration exists in the attic and antrum; second, in carionecrosis of the lateral attic wall; third, in cholesteatoma and polyp formation involving the attic and antrum; fourth, in caries of the tegment tympani; fifth, in carionecrosis of the promontory; sixth, in cases of middle ear suppuration without involvement of the mastoid when symptoms of complication of the inner ear and brain appear. The mastoid operation may be done in: first, carionecrosis of the posterior osseous wall of the canal; second, in chronic suppuration with cholesteatoma of the mastoid cells, though Kashiwarbara believes that his method of atticoantrotomy will suffice in a large number of these cases. He says the radical operation is indicated: first, in extensive suppuration of the mastoid; second, external fistula leading into the mastoid; third, changes of the posterior wall of the mastoid cavity which often lead to brain complications; fourth, disease of the lateral sinus; fifth, necrosis of the internal ear. He claims that by his method of operating, paralysis of the facial and tinnitus aurium appear much less frequently than after the radical operation, and with skilled operators practically are never seen. *Wood.*

II.—NOSE.

Contribution to the Anosmia of Firemen.

O. Levenstein (*Archiv. für Laryngologie,* Vol. 25, No. 3) reports two cases of firemen who experienced partial loss of smell after heavy blows on the head, which developed later to a more complete anosmia, a condition which he attributes to severe mental and bodily strain undergone by the individuals in the course of their calling. *Goodale.*

Triangular Opening of Maxillary Sinus from the Nose.

E. Richter (*Archiv. für Laryngologie,* Vol. 25, No. 3) describes his operation as follows: Chiseling the nasal wall of the sinus below the turbinate, sawing with the double saw

chisel from the interior of the sinus towards the nose, breaking off the oblong, window-like corner of the bone. Subsequent treatment is by tamponing and irrigation. The Killian-Klaus chisel is used. *Goodale.*

Endothelial Tumors of the Nose.

POLLAK (*Archiv. für Laryngologie,* Vol. 25, No. 3) reviews the literature and reports a case, coming to the conclusion that their origin, whether endo- or epithelial, is not yet solved. Their morphology speaks in favor of an endothelial origin. They may be grouped under the head of organoid and mixed tumors of the nose and sinuses, or presumably endothelial origin of the parenchymatous cells. The socalled lymphango-endotheliomata belong to the first or organoid class, the mixed tumors after the type of the salivary tumors belong to the second class. The cylindromata are to be reckoned with the organoid or the mixed tumors, according to their histologic constitution. *Goodale.*

On the Production of Tone in the Nose in Speech and Song, and on the Occurrence and Significance of the Passevant Swelling.

FROESCHELS (*Archiv. für Laryngologie,* Vol. 25, No. 3). The growth or prominence described by Passevant, represented by the muscular apparatus of the posterior pharyngeal wall, supports the palate in its work of closing the naso-pharynx, and according to this author occurs only in nonnasal sounds. The author shows, however, by radiograms that this prominence can also occur in nasal sounds, and that it is, therefore, their function besides affecting complete closure. The author shows, further, that the nasal tone is dependent upon the spoken or sung vocal, and consists of an irregular relation between the strength of the nasal tone and the length of the lower air tube. The strength of the nasal tone is directly proportional to the completeness of the palatal closure. With increased vocal strength the nasal tone diminishes under physiologic conditions. The height of the tone influences the strength of the nasal tone. *Goodale.*

Inflammation of the Antrum in the Newborn.

CANESTRO (*Archiv. für Laryngologie,* Vol. 25, No. 3) reports the case of a child 26 days old, which showed ten days

after birth reddening and swelling of the infraorbital region, with exophthalmus and purulent discharge from the same side. A fistula was found in the mouth over the upper alveolar margin, which was incised and two teeth removed. Recovery followed in two months. Examination of the pus showed staphylococcus aureus. Nine other cases are found in the literature. The condition is to be distinguished from ocular lesions with extension to the inner angle of the eye, from lesions of the nose, such as simple purulent discharge in coryza and rhinitis, from lesions of the teeth with swelling or fistula on the margin of the gum or the palate, from renal disease or anemia, with temporary edema of the face, and from acute infectious diseases where the only symptom is fever.

Goodale.

Complications of Maxillary Empyema.

PAUNZ (*Archiv. für Laryngologie*, Vol. 25, No. 3). In this condition the majority of cases show an accompanying periostitis, which leads to extensive necrosis of the maxillary walls in the nasal process, since often in the beginning there exists periostitis of the alveolar process. Where this is not apparent to the eye, there certainly exist in the bone tissue periosteal inflammations, from which a further extension of the inflammatory process is easily possible. These severe complications probably are due especially to virulent bacteria. While in the majority of cases these present mild infections and a good prognosis, it is possible, on the other hand, that they may lead to complications of severest severity. From the prophylactic standpoint it is extremely important to give the greatest care to the upper bicuspids and molars, and one should be very guarded with reference to conservative treatment of these teeth. The filling of these teeth is apt to bring severe inflammation of the maxillary sinus, especially when a pulpitis is overlooked. *Goodale.*

The Comparative Merits of the Various Methods Employed in Operations for Septal Spur.

KANTER (*New York Medical Journal*, October 28, 1911) regards the following as among the chief advantages for the removal of spurs by submucous resection:

1. It avoids troublesome secondary hemorrhage by leaving no exposed cut surface of cartilage or bone.

2. It takes very little, if any, more time than the saw operation.

3. There is no bothersome scab and crust formation.

4. We leave a functionating mucous membrane in place of scar tissue that would result from the saw operation.

5. There is no recurrence of the spur from thickening of the exposed cartilage or bone and scar in the mucous membrane.

6. The wound in the mucoperichondrium heals rapidly. In five days or a week healing is complete. *Harris.*

The Treatment of Nasal Synechias.

BRINDEL (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* July 8, 1911) recommends as superior to the introduction of separating materials, the application of crystals of chromic acid, after thorough separation of the synechia, to the two bleeding surfaces. The application of the chromic acid is to be immediately followed by a copious nasal irrigation, made through the nostril on the opposite side. The wound is left entirely alone for five or six days, at which time the scab made by the chromic acid will fall off.

Harris.

An Anatomic Study of Regional Anesthesia of the Superior Maxillary Nerve.

BARIL (*Revue Hebdomadaire de Laryngologie,* September 2, 1911). The nerve supply of the antrum of Highmore, according to the author, is derived from the branches of the superior maxillary nerve and from Meckle's ganglion. The nerve and the ganglion both are situated in the posterior portion of the pterygomaxillary fossa, near its floor. Local anesthesia of the terminal branches of the nerve, as proposed by Gordon King and others, has been only partially satisfactory. As a surer method, anesthesia of the nerve itself in the pterygomaxillary fossa has been proposed by several authors. The writer considers the various channels of approach, and rules out the external route through the cheek and the orbital route in favor of the approach through the inferior buccal along the posterior palatine canal, which is from 2 to 3 mm. in diameter at its smallest portion. Examination of the canal upon the skull shows, first, that the buc-

cal opening is situated at the base of the third molar tooth; second, that its direction is slightly oblique across the neck of the second molar tooth; third, the nerve will be found regularly at a depth of 4½ cm. from the neck of the second molar tooth.

He proposes the following technic: The mouth is opened to its fullest extent; a platinum needle 5 cm. in length is introduced into the gum for about 4 mm. inside the middle of the neck of the second molar. The barrel of the syringe rests upon the lower lip and is carried forward for about one cm. until it engages the head of the third molar. Here the posterior palatine canal is always met. The important point for finding the nerve is not to carry the needle in too oblique a direction upward and backward; otherwise the needle will fail to enter the canal, and glide along the wing of the pterygoid plate, perforating the mucous membrane of the pharynx. This is shown by the fluid escaping into the throat. It is advised not to introduce the needle more than 5 cm. at the most; 4½ cm. will usually suffice. The author believes that there is no danger of septic involvement, and that a possible wounding of the small palatine artery is of no importance. For the operation he makes use of the following formula: Hydrochlorate of cocain, 5 cg.; novococain, 10 cg.; adrenalin, 1/1000 solution, 20 drops; normal saline solution, 10 cg. He injects 3 cubic cg. of the solution in the posterior palatine canal, and up to 5 cm. in the depth. Ten minutes after the introduction, anesthesia will be perceived in the lip, with the sensation of swelling in the region. Twenty minutes later he advises a submucous injection of the same solution as an additional precaution. The operation can then be done without the slightest pain. He intimates that he has not used the method in many cases, and is not prepared to state the actual results as yet. *Harris.*

Consideration of the Treatment of Atrophic Rhinitis With Crusts, With or Without Ozena, by Alkaline Vaporization, Followed by Vibratory Massage of the Mucous Membrane of the Nose and Nasopharynx.—Results of Five Years of Employment.

SIEMS (*Revue Hebdomadaire de Laryngologie,* October 21, 1911). Atrophic rhinitis, according to the author, is a morbid entity in which the clinical picture varies according to the

circumstances, the area in which it developed, determining cause in the last analysis, and according to the stage of its evolution. Two extreme types are to be recognized: one where the process is limited to the erectile tissue and is characterized by a formation of crusts, most often in the back of the nose; the other form where the bony substratum is involved and the mucosa is markedly reduced in thickness.

Here a new symptom is added—that of odor. Between the two types one meets a multitude of variations. Siems recommends for the removal of the crusts a hot alkaline vaporization, temperature 40 to 50 degrees C. (Vichy water), under a pressure of two to three atmospheres. For its effect upon the mucous membrane he follows this up by a vibratory massage by means of an electric motor, making use of ichthyol or balsam of Peru. Ichthyol, in his opinion, is far superior to the balsam of Peru, the chief advantage of the latter being its pleasant odor. By the action of the vapor the crusts are softened and partially liquefied. Another effect is the "tempering" of the mucous membrane. The alkaline solution forms a soap with the oily materials in the accessory sinuses, and also in the cavities of the gland, formed by the destruction of the acini. The radioactivity of the Vichy water has an additional beneficial action. The neutralization of the acids arising from the fatty materials and the secretions of bacterial origin in the Schneiderian area is produced. Its action is particularly beneficial upon the bony substratum. The atrophic process here is in large part due to the solution of the phosphate of soda by the action of the carbonic acid and the lactic acid, produced by the bacteria, and resulting in the decomposition of the putrefied material in the nose. During the vaporization the patient ejects large quantities of this putrid material from the nose and at times from the throat. It is at times necessary to remove the crusts mechanically, but it is never required to use the probe. The author believes that mechanical massage has decided advantage over the method of Braun, which is at the same time painful and not so effective. He goes at length into the physiologic result of massaging the healthy mucous membrane of the inferior turbinal bone, which is to excite the cavernous tissue, dilating the vessels and the excretory canals of the glands, which deposit their contents upon the surface of the mucous membrane.

When massage is applied to the atrophic mucous membrane, two new phenomena are noted. The first, the secretion, which is wont to appear at the mouths of the glands, is yellow, thick as honey, and gives forth an odor of sebaceous material, similar to that met with in ozena. The nasal fossæ, which have been thoroughly cleaned, are again found to contain secretion above and behind. In a word, the vibratory massage expresses the acini and empties them of their putrid contents. He does not deny that the technic is difficult and liable to be painful. He applies it both to the nose and to the nasopharynx, where it is especially difficult. A gentle motor and a good sound are essential. By its action the mucous membrane is uniformly congested, and this congestion is accompanied by abundant exudate and, in consequence, a migration of leucocytes. The patients have the sensation of having caught cold. The first effect of vibratory massage is, then, to promote healthy vasomotor action by the excitation of the terminal nerve filaments, and next to stimulate the muscular tissue, in that way bringing the blood to the part and ultimately increasing the deposit of phosphate of soda. Its beneficial action upon the ozena is decided. Siems summarizes his results at length, claiming to have cured fifty out of sixty cases where the method was employed. The patients were from all parts of the world. Among them were several children. The application of the treatment here is admitted to be difficult.

Not bearing directly upon the treatment under consideration, but of interest, are his observations of the frequency of chorea in his cases—twelve times in twenty cases. Hypothyroidia was met in ten cases out of twenty-three, all women. He pertinently inquires whether there may not exist as a cause of chronic atrophic rhinitis, some disturbances of the internal gland secretion, either hereditary or congenital.

Harris.

Preliminary Notes Upon the Radical and Esthetic Cure of Frontal Sinusitis by Means of Beck's Paste.

Torrini (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* December 9, 1912) has experimented with the use of Beck's paste after the removal of all disease, to overcome the cosmetic defects which are wont to follow operations upon the frontal sinus. His method is to make a

thorough exposure of the sinus externally, preserving the periosteum in its entirety, then to remove all the anterior wall and thoroughly curette away the diseased mucous membrane. A wide opening is made into the nose. He then introduces, under the strictest aseptic precautions, the formula for Beck's paste. He brings the periosteum together by fine silk sutures, and the skin by a separate set of sutures, employing a collodion dressing outside. He has performed the operation upon four lambs. In none of the cases was there any septic after results. Two of the lambs were killed shortly after the operation, the other two at an interval of a year. In one case, due in his opinion to the fact that a dead space was left, there was some suppuration which made its way externally. In all of the cases there were most perfect cosmetic results. In examining the animals killed at the end of year, it was impossible to find in any of them the incision in the skin, or the area where the operation had been performed. The paste was found in situ, undisturbed. He feels that the method can be employed without fear of the paste breaking down through the invasion of septic matter from the nose; indeed, that it forms a barrier against such invasion. *Harris.*

The Surgical Treatment of Frontal Sinusitis—A Critical Study of the Postoperative Sequelæ.

Sieur and Rouvillois (*Archives Internationales de Laryngologie d'Otologie et de Rhinologie*, May to October, 1911), consider in an exhaustive way the various complications following operations upon the frontal sinus, whether by the intranasal or the external routes. A complete perusal of the article is essential to its full appreciation. The subject is one of great interest to every rhinologist.

The authors regard as postoperative complications, accidents which appear clinically immediately after the operation, or so soon after as to be logically associated with the operation. Two categories of cases are excluded: those in which the intervention shows that although the complications were not recognized clinically, they none the less exist anatomically prior to the operation (such are cases of osteomyelitis, extradural abscess, cerebral abscess, etc.); second, cases in which, while clinically symptoms were absent prior to operation, the operation itself did not give any explanation for the resulting

trouble which developed so suddenly that it could not possibly be ascribed to the operation.

Their study of the subject is considered under six headings: first, history of operations upon the frontal sinus; second, a résumé of the histories of postoperative complications as given in current literature; third, the etiologic conditions leading to these complications; fourth, the channels of infection; fifth, prophylaxis; sixth, conclusion.

The authors at the outset deplore the scantiness of the literature in spite of the certainty of a large number of such cases. In Chapter II they analyze the various cases reported under the two heads of "Complications Following Intervention by the External Route," and "Complications Following Intervention by the Endonasal Route." Of complications following intervention by the external route, they have been able to collect 71 cases, divided as follows:

Hemorrhage	2
Orbito-ocular	17
Osteomyelitis	17
Thrombophlebitis and septic pyemia	2
Mild meningeal accidents	3
Severe meningeal accidents	27
Brain abscess	3

Postoperative complications, involving the orbit, are far less frequent than the spontaneous complications which are observed in this region. Of the 17 observations, the most important were diplopia, paralysis of the superior oblique, 7 cases; phlegmon of the upper lid, 3 cases; phlegmon of the orbit, 1 case; optic atrophy, 2 cases; optic neuritis with pyemia and thrombosis, 1 case. They feel that the danger of diplopia following operation on the frontal sinus has been much exaggerated. In their operations they have never met with the accident. Care in the preservation of the periosteum in the region of the superior oblique will cause this accident always to be avoided. In the author's opinion the infrequency of orbital infections is remarkable. This was ascribed to the resisting power of the periosteum.

Osteomyelitis. The critical study of the 17 cases reported strongly emphasizes the multiplicity and the long duration of the lesions of the accessory sinuses in the cases

operated upon. As a result of this, certain anatomic lesions special to osteomyelitis are to be noted in the cases reported. The ethmoiditis is characterized by a rarefying osteitis, and the entire bone is easily removed by means of a curette. The region of the middle meatus is obstructed by a mass of polyps through which the pus drains with difficulty. As a result, headache is a common symptom. Because of the connection between the vascular system in the mucous membrane and that in the bone, a rarefying suppurative osteitis develops in the walls of the frontal sinus, characterized by an accumulation of embryonic cells and of leucocytes, which produce, in time, granulations in the bony canaliculi. This in time leads to necrosis and to the perforations commonly met with in the inferior or orbital wall, or upon the anterior frontal wall. The thinness of the walls is such, however, that soon the suppuration forces its way to the surface, and there results a formation of subperiosteal or subdural abscesses. Another peculiarity of the disease results from the structure of the frontal sinus. The continuity of its tissue with the bony tissue of the rest of the cranium produced by the periosteum and the dura mater on the one hand, and especially by the bony canaliculi containing the plexuses of veins and of plexuses on the other, allows an uninterrupted extension of the inflammatory process to the parietal, temporal and occipital bones, and even to the bones at the base of the skull.

Thrombosis, phlebitis, and septic pyemia, of which two cases are reported. It is usually a complication of osteomyelitis or of meningitis.

Meningitis. Thirty cases reported, of which 3 recovered and 27 died. Luc's division of meningitis is followed, namely: (a) subacute leptomeningitis, rapidly becoming general, or fulminating in postoperative meningitis; (b) meningitis more or less circumscribed; (c) serous meningitis. Of the 30 cases above reported, 17 are to be assigned to the first division, because of the rapid appearance and progress of the disease; to the second group belong two cases of Castax developing on the fifteenth and twentieth day after the operation. Usually this form is characterized by a gradual advance, and if the virulence of the germs is attenuated, and the general condition of the patient permits, recovery can be looked for. Only 3 cases of serous leptomeningitis appear in

the series, all characterized by the benignancy of the disease. All recovered.

Brain Abscess. The authors agree with Luc that the development of disease of the brain in the frontal region is in the great majority of cases entirely independent of all operation, and is dependent upon the extension from the already infected sinuses. Of the 36 cases reported by Luc of brain abscess, he was unable to demonstrate the postoperative origin of the abscess in more than 10 cases, and even in these it was not certain that in some of them the infection did not precede the operation. Of the complications following operations upon the frontal sinus through the nose, the authors have little to say, except to insist upon the frequency of their occurrence.

Chapter III is devoted to the etiologic conditions predisposing to postoperative complications. These are considered first from the standpoint of predisposing causes which are inherent in the patient, and second, of predisposing causes in the operator. The surroundings in which the patient is operated upon, in the opinion of Coakley, is of considerable importance. The young are far more exposed to osteomyelitis than the old. Of the 17 cases reported, 9 were found in patients under fourteen years of age. The general condition of the patient is of great importance; a patient suffering from diabetes, from bronchitis, or Bright's disease, aged individuals, all are liable to postoperative complications. In the same way, individuals suffering from pansinusitis, and those with fever and headache of long duration, all call for the avoidance of an operation. Syphilis, tuberculosis, and the infectious diseases all appear as etiologic factors. As local conditions favoring postoperative complications are to be mentioned the anatomic formation of the sinus. The authors distinguish three forms: small, large, and medium size sinuses. In the first form one is most liable to enter the cranial cavity. Clinically, the large sinuses are much more dangerous, from the standpoint of complication, than those of medium size, inasmuch as the walls are wont to be particularly thin. They also present very often dehiscences upon the orbital or cerebral walls. In such cases the orbital periosteum or the orbital meninges are in direct contact with the mucous membrane of the sinus, and infection by propagation can take place

without any barrier of bone to intervene. Moure and Grunwald have called attention to the narrowness of the region in front of the nasopharyngeal canal, when the septum is found deviated toward the os planum. In such cases the canal at the close of the operation is found to be so narrow, as a result of the swelling of the mucous membrane, that it is virtually obliterated. The medium sized sinuses are especially met with in osteomyelitis. The mechanism of these infections has been carefully studied by Breschet. In certain subjects, particularly in young females, there exists between the two compact layers of the anterior wall of the sinus a diploic layer which is sometimes 7 to 8 mm. in thickness. In this diploic tissue Breschet has described a vascular plexus running in the various bony channels which open on the one end in the cutaneous surface and on the other in the meninges. Its plexus is not limited to the frontal bone, but extends over the parietal, temporal and occipital. The veins which are present here have connection with those of the brain, the meninges, the orbit, and of the frontal sinus. This easily explains the rapidity of the infection which takes place in cases of osteomyelitis, as well as the development of subperiosteal and subdural abscesses.

2. The extent and depth of the lesions of the frontal sinus and the neighboring cavities are important etiologic factors. Isolated frontal sinusitis is a rare lesion. Almost always it is associated with anterior ethmoiditis, and very frequently involvement of the maxillary sinus. Frequently, too, the intersinal septum shows dehiscence in the presence of pansinusitis. Incomplete operation is almost certain to lead to postoperative complications. Finally, the condition of acute local infection, or chronic infection with an acute exacerbation, is a contributing factor.

3. The nature and virulence of the infection enters into the etiology.

Of causes adherent in the operator are to be mentioned, first, patients where a traumatism has resulted in neighboring structures. This can take place as a result of the mere use of the chisel and mallet. Second, infection as the result of faulty asepsis from the immediate closure of the external wound. In the first case, infection of the meninges can only be produced by the existence of suppuration in the neighboring

sinuses, as in cases reported by Luc and Killian. In the second, the infection is produced indirectly as a result of the immediate closure of the skin wound, as in cases reported by Heine and Luc. Third, incomplete operation is a distinct cause of postoperative infection.

4. The Routes of Postoperative Infection. The infection may be either by continuity or conveyed from a distance, either (a) through the venous system, or (b) through the lymphatic system. Hajek admits infection by continuity for all the accessory sinuses of the uose, and particularly for the frontal sinus. In his opinion the initial infection commences in the mucous membrane, giving rise to a subperiosteal abscess ending in necrosis of the bone. The necrosed bone gives rise to pathogenic bacteria which in turn produces an extradural abscess, resulting in meningitis, cerebral abscess, or a thrombosis of the longitudinal sinus. The characteristic feature of this form of propagation is the slowness with which the infection his spread. On this account the authors believe it occurs chiefly among spontaneous complications rather than among complications postoperative, in time, which are characterized by their rapid evolution. Guisez is of the opinion that in this way is to be explained the pathogenesis of osteomyelitis of the skull. Without denying the possibility of infection by continuity, the authors believe that in numerous cases where it is said to be the method of development, in reality the venous channels through thrombosis of the veins of the mucous membrane and the bone represent the first stage of the more extensive complications. They base their opinion upon the fact that while the size of the venous channels in the diploic skull is very small in the infant, nevertheless, no histologic examination has shown that they do not play a rôle, perhaps an important one, in the transmission of pathogenic germs. They are of the opinion of Hajek, that a frequent mode of propagation of the infection consists in the invasion of the diploic through a thrombophlebitis of the veins in the bone, and they raise the question whether in reality there exists but one channel for the carrying of the infection, namely, the vascular route. However this may be, the infection by the vascular route in the pathogenesis of postoperative complications occupies a position of first importance. According to Killian, the deep venous plexuses in the mucous membrane anastomose

in numerous places with the intraosseous veins. In the bone there exists a delicate vascular plexus, at times visible, between delicate bony laminæ, such as the lamina papyrycea of the ethmoid, or of the septa which separate the ethmoidal cells. The vascular plexus anastomoses at numerous points with the veins of the dura mater, those of the periosteum of the orbit and those of the nasal fossæ. There exist in this way blood channels going from the mucous membrane of the accessory sinuses through the osseous veins into the opposite periosteum and into the dura mater. These channels are often long, complicated, and fine, but at other points they are short, and large. These immediate passages of a mucous vein to a meningeal vein explains to us the possibility of the extension of the infection from the mucosa to the dura mater. The bone in no way forms a septum between the two. It serves to limit the vascular systems, but does not in any manner suppress them, any more than the mountains suppress the relation between two countries, because there exists always numerous passes. It is to be recalled that spongy tissue is particularly developed in the anterior wall at the level of the frontal boss, where it unites with the inferior and posterior walls. This can easily explain the development of an osteomyelitis. The examination of the relations which exist between the veins of the mucous membrane of the frontal sinus and the intracranial surface is not less interesting. Some empty directly into the meningeal veins and by their intermediation into the superior longitudinal sinus. The others are tributary to the ethmoid veins, and anastomose upon the cribriform plate with the veins of the dura mater of the superior longitudinal sinus. This anastomosis is made by two routes: one a direct, the other by a devious route, analogous to that which we have just studied; while those which follow the direct route empty into veins with small orifices lying in the posterior wall of the frontal sinus, at times visible to the naked eye, and capable of permitting the introduction of a fine probe. Those which follow the devious route are those which take part in the vascular interosseous system described by Killian. The anastomosis of the venous circulation of the mucous membrane of the frontal sinus with the circulation of the orbit is of a similar nature. The anatomic relation of the venous systems proceeding from the mucous membrane of the frontal sinus

serves to explain why the propagation of the infection can vary in rapidity according to the case; gradually by infection by the intermediation of the interosseous systems, rapid and fatal in action where propagation is through the direct transosseous anastomosis. The first mechanism serves to explain those cases where the complications have developed gradually and have followed, apparently, in appearance, the route called continuity. The second will apply to cases in which the operation served in the course of forty-eight hours to set up a fatal subacute meningitis.

For an accurate statement of the method of infection, microscopic examinations of the tissues of the mucous membrane of the frontal sinus and the brain are essential. Up to the present time this has not been practiced, except in three cases. One of these is the case of Hajek, a young girl who had been treated by him for over a year by the intranasal route, and who suddenly developed meningitis, and, in spite of an intracranial operation, died the next day. The autopsy showed acute purulent pachymeningitis, with leptomeningitis at the base of the skull. The histologic examination showed the deep layers of the ethmoid mucous membrane the seat of an intense congestion with hemorrhagic areas. The bacteriologic examination demonstrated that the mucous membrane contained streptococci which had penetrated into the interior of the intraosseous veins, and from there into the meninges. In addition to the infection by the venous system, it has long been held that infection may proceed through the lymphatic system. Much remains to be done in the way of accurate scientific work to demonstrate that this is the case. d'Axel Kéy and Retzius have demonstrated the existence in dogs and guinea pigs of direct communication between the subarachnoid spaces and the lymphatics of the mucous membrane. Marc André has shown that this disposition exists also in man, but does not extend beyond the olfactory region of the mucous membrane. Falcone, as a result of his anatomic investigations on the subject, has arrived at the conclusion that there exists in the mucous membrane of the frontal sinus a lymphatic system which is easy of demonstration, and as a result of an injection of the meningeal spaces through the frontal sinus, it is possible to demonstrate a lymphatic transosseous communication existing between the cranial cavity and the frontal sinus.

Mouret, on the other hand, does not believe that this is possible, and concludes as a result of his studies that in addition to propagation by contact and through the venous system there is a propagation not by the lymphatic system but through a microbic diffusion. At this stage of the discussion, in the authors' opinion, no definite conclusions can be formed as to the certainty of the transmission by the lymphatic route.

5. The prophylaxis of postoperative complication. This is considered under the heads of, first, the choice in the operative procedure; second, the preparation of the patient; third, the execution of the operation.

As regards the choice of the method of procedure, they recognize the claim of Hajek, Tretrop, and others that many cases only require intranasal work. At the same time, in their opinion, we must not conclude that it is the method of choice, because the execution of a systematic endonasal method is not free from dangers, and a large number of cases regarded as cured are wont immediately to relapse upon the first serious attack of a cold. On this account, while they recognize the necessity of making a faithful attempt of the method in chronic cases, they believe that its too prolonged application in patients is unwise and attended with dangers. They admit that here, as in all their cases, it is necessary to avoid extreme formula when the truth rests in a happy medium.

Preparation of the Patient. Here a careful examination of the patient's general condition is important. This is too often neglected. Further, every means should be taken to determine the extent and nature of the disease. Under the helps, in the authors' opinion, but of restricted value, in this direction, is the radiograph. In the execution of the operation itself attention should be placed upon the importance of doing any endonasal work preliminary to the external operation.

Two groups of cases, as far as complete intervention is concerned, are met with in performing the external operation. In the first group the operation should be as brief and limited as possible. It includes, 1, cases of inflammation of the frontal sinus with acute exacerbation; 2, certain pansinusites; 3, advancing osteomyelitis. In frontal sinusitis with acute exacerbation, only the simplest procedures should be undertaken until the acute stage has subsided, and then only when there is rapid evolution of the disease with a severe pain, which can-

not be controlled, due to the retention of pus, with high fever; rapid swelling of the frontal region, with the formation of a subperiosteal abscess, indicating an osteomyelitic process; signs of meningitis. In the second class, certain pansinusites; the patients here are subjects of extensive lesions, and have their digestive and respiratory tracts infected by the penetration of the products of septic secretion. In these conditions it is essential, before undertaking the radical operation, to disinfect the sinus and remove by the endonasal route all the disease possible. Later, if the general health permits, and there is no other objection, a conservative external operation can be undertaken. Third, osteomyelitis. As a result of the authors' observations they are of the opinion that we are entirely powerless to combat any advance of osteomyelitis. In the 17 cases reported, death followed sooner or later, in spite of all surgical intervention. Contrasted with the cases where the intervention should be avoided, or limited in nature, are the cases where it should be complete. While a complete operation is essential, undue violence in the operative procedures is to be condemned in the removal of the bony walls, during the curettage of the frontal sinus, or during the curettage of the ethmoid. Second, the importance is to be emphasized of the necessity of the maximum amount of drainage, whether through the operative wound or by the frontonasal canal. Luc, Killian, and others insist strongly upon the importance of this, and numerous clinical cases justify their insistence. In a great number of their observations they have traced the origin of the postoperative accidents to insufficient drainage. *Harris.*

III.—PHARYNX.

A Peculiar Recurrent Mycosis of the Tongue.

GANTZ (*Archiv. für Laryngologie*, Vol. 25, No. 3) reports a case characterized by a slight hyperemia of the surface of the tongue, with prominence of the papillæ. Later a light gray and very thin coating appeared on the surface, which increased rapidly in thickness, simultaneously penetrating into the depths. The covering at the beginning was firmly adherent, but later gradually exfoliated, leaving a bare area which rapidly took a normal appearance. The disease was characterized by the occurrence of numerous fungous islands,

consisting of thin, symmetrical threads, extending radially from the center to the periphery and not staining by Gram.

Goodale.

The Treatment of the Phlegmonous Inflammations of Waldeyer's Ring.

E. WINCKLER (*Deutsche medizinische Wochenschrift,* No. 46, 1911). The phlegmonous inflammatory processes of the lymphatic ring of the pharynx are always preceded by acute infections of the whole or the larger part of the ring. The abscess develops later in some part of the adenoid substance. Bacteriologic examinations of the pus showed mainly streptococci, more rarely staphylococci. Retropharyngeal abscesses frequently developed after scarlatinal anginas.

In one case of deep abscess of the lingual tonsil the infection probably resulted from a carious molar of the right lower jaw, while in two other cases there had probably been an injury of the lingual tonsil. Both these patients stated that they had been chewing a blade of grass which they had pulled out. In the majority of the cases, however, the etiology was very obscure.

The following forms must be distinguished: First, abscess of the faucial tonsil, peritonsillitis; second, abscess of the posterior wall of the pharynx, the retropharyngeal abscess; third, abscess of the lingual tonsil or abscess of the base of the tongue.

Bilateral abscess of the tonsils and peritonsillar abscess are relatively rare. The combination of retropharyngeal abscess and peritonsillar abscess is also uncommon. The author has only seen one case. Anatomically there is a great difference in the tonsils in which repeated peritonsillar abscesses had occurred and in the chronically diseased tonsils which were removed for other causes. In the simple chronically diseased tonsils the capsule covers the tonsil uniformly, while in the tonsils in which suppurative processes had occurred the capsule is broken through at different points by tonsil tissue.

The idea that peritonsillar abscesses originate only in the upper part of the tonsil is not correct. The pus is found almost as frequently laterally and low down; in fact, the whole tonsil may be surrounded by an abscess. Patients who have had one attack of peritonsillar abscess are very suscept-

ible to further attacks. This fact and the difficulty of keeping up good drainage after incising the abscess is an argument for the removal of the tonsil during the attack. It is much easier to remove the tonsil during the height of the attack—and this is particularly true of the small submerged tonsils, whose removal under normal conditions is often a very difficult operation.

When the pillars of the palate are infiltrated and edematous, it is easy to dissect out the tonsil with the capsule and the extracapsular tonsil tissue which has been loosened by the pus, so that adhesions with muscular tissue can easily be separated. The operation, which is done under general anesthesia, is performed in a short time and affords the patient prompt relief and protection against further attacks. Unless a complete tonsillectomy is performed, however, the operation will not prevent recurrences, and is absolutely useless.

In retropharyngeal abscesses the infection of the pharyngeal tonsil plays the most important rôle. Abscesses localized only in the pharyngeal tonsil are rare. More frequently the infection extends to the loose connective tissue between the pharyngeal fascia and the prevertebral fascia of the neck. The danger of such an infection in childhood consists in the extension of the suppurative process along the anterior surface of the vertebræ to the fifth or sixth cervical vertebræ and posterior surface of the larynx, causing great difficulty in breathing.

The treatment in such a case consists in a crucial incision through the abscess wall on the posterior wall of the pharynx, just as soon as the condition is recognized, thus preventing a downward extension of the infection. If the pus has extended so deeply that pressure against the larynx is being produced and swelling of one side of the neck, an external operation must be performed. The lateral pharyngeal wall can be reached by an incision along the anterior border of the sternocleidomastoid muscle. If possible a tracheotomy should be avoided, because it paves the way for further infection. The difficulty in breathing stops as soon as the abscess is opened.

Suppurative conditions of the lingual tonsil may be merely tonsillar abscesses which cause swelling at the base of the tongue, or the muscular substance of the tongue itself may be involved. In the latter infection, August Killian has pointed

out that an incision between the two genioglossi or between the genioglossus and hyoglossus is the best treatment.

In conclusion, the author states that in phlegmonous inflammations of the lymphatic ring, incisions in the pharyngeal cavity are not sufficient. The radical operation, tonsillectomy, does not only remove the existing disease, but prevents recurrences of the same condition. *Theisen.*

IV.—LARYNX.

Laryngeal and Tracheal Carcinomata in Association with Carcinoma of the Esophagus.

ZIBA (*Archiv. für Laryngologie*, Vol. 25, No. 3) reports two cases which show that the combination of the two growths which originally appeared to arise in the multiple form, representing, nevertheless, a genetic relationship in which the carcinoma of the esophagus was secondary. The structural difference between primary and secondary tumors arose through anaplasia, prosoplasia or metaplasia. The influence of external conditions of life, which vary according to the organ, exercises marked difference on the tissue cells. The esophagus possesses conditions well adapted for cancroid formation, so that other types of carcinoma can be transformed by prosoplasia or metaplasia into cancroid. *Goodale.*

Surgical Treatment of Laryngotracheal Stenosis with Special Reference to the Translaryngeal Method of Fastening the Drainage Tube.

SCHMIEGELOW describes a method which he employed in a case of laryngeal stenosis following attempted suicide. The patient exhibited a fibrous adhesion of the ventricular bands, and had breathed through a tracheotomy tube for six months. The vocal cords were normal. The ventricular bands were adherent by cicatricial tissue, which was removed under chloroform anesthesia, and a gutta percha tube five centimeters long, with thick walls, corresponding to Chamieres Filieres No. 39 was introduced, and was fastened with silver sutures, passing transversely through the larynx and the tube. Five weeks later the drainage tube was removed by local anesthesia through the mouth, and eight days later the tracheotomy tube

was removed, as the larynx was sufficiently healed. Respiration was quiet during sleep, but restricted during violent exertion, and a second tube was introduced in the same manner, which he wore for four weeks, when it was removed and the passage made entirely free. *Goodale.*

Cicatricial Stenosis of the Lower Portion of the Trachea—Section—Dilation.

LAURENS (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* August 5, 1911) reports a case occurring in a man of 33, who was brought to him in a state of intense dyspnea. Tracheoscopy under cocain showed at the lower end of the trachea, about 2 cm. from the bifurcation and more than 20 cm. from the teeth, a cicatricial diaphragm with a punctiform opening measuring scarcely 4 mm. in diameter, through which an air current passed with difficulty. The orifice was slightly eccentric. The diaphragm was composed of a membrane thin enough to vibrate with each passage of air. This membranous portion was surrounded by a thicker cicatricial ring, which gave to this region of the trachea the appearance of infundibula. The author was able by means of a manderin of 7 mm., introduced through the tracheoscope, to dilate the orifice. On subsequent visits he was able to introduce a manderin of 9 mm., and later incised the cicatricial ring by means of a uretrotome, which permitted the introduction of a manderin of 11 mm. The patient has been kept under observation, and there has been no return of the cicatricial condition. The cause of the obstruction is not clear. The patient, however, gave a history of syphilis. *Harris.*

Five Cases of Laryngostomy.

SIEUR AND ROUVILLOIS (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* August 12, 1911) report five cases of laryngeal stenosis treated by laryngostomy, and draw the following conclusions from them:

First, the benignancy of laryngostomy, provided that it is not performed while an active inflammatory process is going on. All the cases were operated upon under local anesthesia. By the proper position of the head and careful sponging, little blood need enter the trachea. None of their cases were

followed by bronchitis, and the evening temperature in none of them for the first two days exceeded 38°. They contrast the operation with the different methods for dilatation of the larynx through the mouth, decidedly to the advantage of the former.

Second, they lay the greatest emphasis upon the time when the dilatation is to be begun, advising no undue haste in this. In one case where the cartilage of the larynx was involved, almost a year was taken for the dilatation. At times it will be necessary to interrupt the treatment because of temporary resection due to using too large a tube, which is the third point that they emphasize, feeling that there is danger here in attempting to overdo it.

Fourth, the closure of the laryngeal wound should not be undertaken too soon. As regards this procedure, the cases operated upon by them were divided into two groups. In the first group there was no inflammation of the cartilaginous framework. Here the procedure of dilatation was a short one, and the closure of the wound was effected very simply. In the second group of cases where the cartilage is involved, and there is a general deformity of the larynx, while dilatation was effected rapidly, there was a sort of aspiration of the soft parts of the neck which interfered with the air entering the larynx and necessitated the continued wearing of the tube.

Harris.

Laryngeal Applications by Instillation Through the Nose.

LAVRAND (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* August 26, 1911) calls attention to the need of special skill for making applications directly to the larynx, and to the fact that there are frequent occasions when the services of a specialist are not available. For such cases, and they are many, he recommends the introduction of the medicine to the larynx by gargling according to the method suggested by Guinier. For this purpose, a small portion of the fluid is taken into the mouth, the head is thrown well back, the jaws are wide open, in order to render swallowing difficult, and the patient pronounces the vowel e-e-e-e. In this way the fluid is allowed to pass well down into the pharynx to be immediately ejected, and the larynx mirror will show it bathing the epiglottis and all of the larynx above the true cords. As a

still more satisfactory procedure, he recommends introducing it through the nose, as first suggested by Mendel. In the quiet state the larynx is open except during speaking, and forms a funnel with the large end above, while the esophagus is closed except during swallowing. Under these conditions the liquid introduced through the nose will flow down the pharynx and arrive easily at the opening of the larynx. The tendency for the esophagus to open can easily be controlled by the exercise of the will power. It is only necessary to have the patient breathe regularly, without any interruption, to overcome this. The author has demonstrated the possibiilty of the method by the introduction of a bland oil colored with methylin blue, which can be discovered upon the vocal cords. *Harris.*

Unusual Forms of Cancer of the Larynx.

MOURE (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* September 23, 1911). It has been known for a long time that malignant tumors springing from the vocal cords of persons well along in years are wont to have a slow progress. They remain entirely quiescent for many years and then take on a rapid growth to the point of a fatal issue within two or three years. There is, however, a group of cases which differ decidedly from this type. Here the microscope does not reveal any malignant involvement, even when the tumor has been examined repeatedly for a term of years, due either to the fact that only a superficial section has been removed, or that the growth only later takes on a malignant form. Moure believes that the latter is the case, although he admits that it is hard to prove. This group of cases offer a far more favorable prognosis for intervention of a more or less conservative nature. Such are the cases where cures have been reported by the endolaryngeal removal of the growth. In some of these cases, however, even a removal by the external route has not proved sufficient, and it is only the removal of the larynx in whole or in part that has secured a permanent cure. Moure has had five of such cases under his care. One of these which he reports was a woman of 42, who had had repeated removals of a papillomatous-like growth upon the left vocal cord by other specialists. When seen by him he regarded it as probably malignant, and performed a thyrotracheoto-

my, removing the growth thoroughly, which was found seated in the right ventricle and upon the upper surface of the vocal cord. The patient made a prompt recovery. The tumor, however, returned, and later it was necessary to open the trachea. More radical intervention was refused. The patient at the end of five years was alive, but the growth had increased to large proportions, and a fatal issue was not far off.

Moure has given up in recent years opening the trachea in such cases, in order to avoid the possibility of a perichondritis, or secondary fistula, and in place of this introduces a specially devised canula through the cricotracheal opening. He has, also, abandoned general anesthesia in favor of cocain infiltration. He speaks enthusiastically of thyrotomy as a method for removal of all difficult growths of the larynx, as well as those of a malignant nature, when limited to one side without peripheral infiltration. *Harris.*

Some Cases of Tumors of the Trachea and Bronchi.

GUISEZ (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie*, October 14, 1911) reports ten cases where he has met with such growths, six of which were benign. The first was a benign tumor of the trachea, diagnosed and removed by the tracheoscope. The second was a papillomatous growth of the larynx and trachea, direct laryngoscopy and tracheotomy. Stenosis of the larynx followed, requiring laryngoscopy. The third was a double intertracheal valve causing severe dyspnea, diagnosed by the tracheoscope and operated on in the same manner, with complete cure. The fourth was a hernia of the posterior wall of the trachea, also diagnosed by the tracheoscope. The fifth and sixth were cases of polypi of the trachea secondary to tracheotomy and removed by lower tracheoscopy. The seventh was a primary cancer of the trachea with secondary involvement of the esoghagus. The eighth was a growth of the posterior wall of the trachea, shown by the tracheoscope. The ninth was a primary cancer of the trachea, diagnosed and treated by the direct method. The tenth was a particularly interesting case, an epithelioma of the bronchus, diagnosed by upper bonchoscopy.

In the author's experience the most frequent forms of be-

nign growths met with in the trachea are fibroma and papillomatous polyps secondary to tracheotomy. Primary malignant growths of the trachea are not rare. They are usually epitheliomas and carcinomas, which grow slowly and reveal themselves first by difficulty in respiration. The author dwells upon the value of the diagnosis and treatment by means of the tracheoscope in such cases, and advises the use of the largest tube that will go through the vocal cords. Preliminary tracheotomy is only called for where the growth is particularly large. Preparation should be made for instant tracheotomy, however, if necessary. Local anesthesia is advised. Guisez is particularly insistent that the tube spatula should be avoided except for examination of the larynx. *Harris.*

Bronchoscopy in Small Children.

KILLIAN (*Vercin Deutscher Laryngologen,* 1911, p. 118). The question which Killian set himself to solve was whether an upper bronchoscopy was ever indicated in a child of six years old or under. He was able to collect from the literature 19 cases where the upper bonchoscopy was done in children under six years, where there was a subsequent subglottic swelling necessitating a tracheotomy. One thing was clear from a review of these cases: before the operations there was no indication to do a tracheotomy, after the removal of the foreign body by the upper route these cases required the procedure as a life saving measure. In 6 of the cases six to twelve hours elapsed before the tracheotomy, in 9 cases twenty-four hours elapsed and in 3 other cases the alarming symptoms did not develop until twenty-eight, thirty and thirty-seven hours after the bronchoscopy. The cause of the trouble in all cases was a subglottic swelling; in a few cases seen with the bronchoscope, in one case observed postmortem.

The ages of five of the cases ranged between the fifth and seventh years, while the other fourteen were between one and four years. He concludes that children of seven and under, but especially children of four years and under, are especially sensitive to manipulation by an upper bronchoscopy and that a dangerous subglottic swelling is very apt to follow. In order to determine the reasons for this condition, a very careful series of measurements were undertaken on a large number of children directly following death and without any harden-

ing of the tissues or other preparation. His very important conclusions briefly epitomized are as follows: The size, or better the width, of the subglottic space bears no relation to the sex of the child and very remotely to the age; the main relation which gives a constant figure is the height of the child. This index is so exact that it is often expressed in half millimeters.

Length of Body. cm.	Diameter of subglottic space. mm.	Circumference. mm.	Age Months.
45	3, 5 (4, 0 refuses)	10.99	8
50	4, 5	14.13	4
52	5, 0	15.7	35
53	4, 5 (5, 0 refuses)	14.3	3
53	5, 5	17.27	newly born
53	4, 5 (5, 0 refuses)	14.13	newly born
56	4, 75	14.91	?
58	5, 0 (5, 5 with pressure)	15.7	2½
58	5, 5 (6, 0)	17.27	4
60	5, 5	17.27	9½
62	5, 5 (6, 0 hard, 6, 5 refuses)	17.27	6
62	6, 0 (6, 5 very hard)	18.24	18
63	5, 5	17.27	9
64	5, 5	17.27	13
65	6, 5 (7, 0 refuses)	20.41	8
70	5, 0	15.7	16
80	6, 5 (hardly)	20.41	12
80	6, 5 (refuses 7, 0)	20.41	24
86	7, 5 (8, 0 refuses)	23.44	48
94	7, 0 (7, 5 refuses)	21.98	36
120	8, 0	25.12	7 years
?	9, 5	29.83	10 years

All measurements were made with steel olive shaped bougies.

This table is given in full on account of its great practical value. The cases were analyzed according to this measurement, and in every case where the diameter of the tube was given, it was shown that too large a tube had been used.

Horn.

Singers' Nodules. Use of Galvanocautery.

C. J. KOENIG (*N. Y. Med. Jour.*, February 11, 1911). For the removal of singers' nodes, Koenig (Paris) makes use of the galvanocautery, which is preferred to cutting methods, as the blood vessels and lymphatics are thus obliterated and the cord protected against any intercurrent or subsequent infection.

The cautery point used is of platinum protected by a small, flat, copper sheath from which protrudes a small ball of platinum, which constitutes the cauterizing part. It is very bright and very easy to see when the reflected light falls upon it. The copper background renders it still more visible. The cauterization by this means can be absolutely localized and united. It is followed by a very slight reaction, a slight redness of the cord, which disappears in a few days.

Richards.

V.—MISCELLANEOUS.

Chronic Pemphigus of the Upper Air Passages.

THOST (*Archiv. für Laryngologie*, Vol. 25, No. 3). The author reports a case characterized by chronic course without fever, simultaneous involvement of the eyes and oral mucous membranes, with normal skin. Gradual contraction occurred of the folds of the mucous membrane of the nasal openings, the soft palate and uvula, eustachian tubes, larynx and epiglottis, and the entrance to the esophagus. The uvula showed in all four cases the same form; likewise the epiglottis where the mucous membrane is in contact showed folds, contractions, shortenings and adhesions. In the eyes four patients exhibited similar appearances, the process in one extending to the mouth, over the lips and reaching to the nose and alæ.

Goodale.

Suppurative Periesophagitis After Extraction of Foreign Body.

JACQUES (*Revue Hebdomadaire de Laryngologie*, November 18, 1911). The patient was a robust man who had swallowed a fish bone, which lodged in the thoracic portion of the esophagus at the level of the bronchoaortic segment, producing immediately excessive and continuous pain, localized in the corresponding area of the spine, with complete dyspha-

gia for all solid food. It was easily removed thirty-six hours afterward through the esophagoscope. Immediate relief of symptoms. Soon afterward, however, the pains returned, accompanied this time by a rapid change in the general condition, fever, sweats, coated tongue, painful cough, etc. Pectoriloquy was detected at the angle of the right scapula. Esophagoscopy was practiced seven days after the removal of the foreign body, and showed the mucous membrane of the esophagus very much swollen, edematous, of a livid color, obstructing and effacing the lumen for several centimeters.

Primary phlegmon of the periesophageal region on the right side and behind the esophagus was diagnosed. Three days later the edema yielded as a result of the evacuation of the pus through the alimentary canal. The perforation was on the level of the left wall, as was shown by the granulation tissue which developed at this point and a drop of pus seen there. Following this there was a rapid improvement in the general condition of the patient, and he made an uninterrupted recovery. The author calls attention to the rareness of such accidents, and that when they occur the issue is generally fatal. *Harris.*

Orthonal—A New Anesthetic.

V. Moses (*Deutsche medizinische Wochenschrift,* No. 46, 1911). Modern pharmacology has shown that combinations of two drugs with similar action frequently have a more potent effect than either used single, while the toxic influence is slighter. These facts have resulted in the desire to supplant cocain with some other less dangerous local anesthetic, because cocain, even when used in small doses, sometimes causes toxic symptoms, faintness, collapse, or even death. When it is combined with another anesthetic it is possible to use a much smaller amount. In this way orthonal originated, a combination of 0.5 per cent cocain with a 0.75 per cent alypin solution to 6 per cent of which a 1-10,000 adrenalin solution is added. This solution is prepared with a physiologic saline solution and sterilized in autoclaves.

Up to very recently orthonal has been mainly used in dentistry. The good results obtained by dentists led the author to employ this anesthetic in minor surgery. During the past two years the author has used it in a great variety of cases in the Friedrichsheim Hospital in Berlin. He also used it for

the infiltration anesthesia of Schleich. One to three ccm. were used each time, and in all cases anesthesia was very complete. No general symptoms, nor bad after-effects could be determined.

It was employed by the author for the following operations: For the removal of atheroma, fibroma, lipoma, nevus, carcinoma of the breast, cysts, isolated hemorrhoids, ingrowing toe-nails, abscesses, for the removal of foreign bodies, needles and bullets, and also for tracheotomies and Winkelmann's hydrocele operation. *Theisen.*

The Lymphatics of the Nose and Nasopharynx, with Consideration of the General Lymphatic System.

HENRY J. HARTZ (*Laryngoscope*, March, 1912) thus summarizes:

1. The lymphatics of the external nose are tributary to the parotid group of glands, the buccal, the submaxillary and the deep cervical.

2. The lymphatics of the nasal fossæ discharge themselves in the lateral retropharyngeal glands and into the deep cervical glands situated under the base of the skull in the region of the middle of the neck. The regionary lymphatics of the pharynx and tonsils are the median and lateral retropharyngeal group, and finally the deep superior cervical, which occasionally anastomose with the lingual and peritracheal.

When we consider the lymphatics of the nasopharynx, we note the following: 1. The richness of the network of lymphatic capillaries and the frequency of the anastomosis everywhere and over the median line connecting both right and left sides of the nose and throat.

The fact that the lymphatic system of the nose and throat does not constitute an entire independency; on the contrary, it is joined with the surrounding lymphatics—those of the forehead, the cheek, the eyelid, the upper lip, the external ear, on one side, and with those of the eustachian tube and soft palate, the pharynx, the sinuses, and the perimeningeal spaces on the other side. Thus the regionary glands belong equally to the neighboring territories. This also is true of the lymphatics of the larynx, thyroid and tracheal bronchial glands. They become tributary to the anterior cervical plexus, which ultimately anastomoses with inferior cervical glands that dis-

charge into the veins the entire lymph collection of the head, neck and upper breast.

3. The lymphatics of the tongue, lips, palate, teeth and floor of the mouth all have intimate connection and discharge into the regional glands, which are the submental, submaxillary and the infraauricular—the posterior part of the mouth joins the lymphatics of the tonsils, and all become tributary to the deep cervical situated along the jugular vein.

4. What interests the clinician and surgeon, especially for purposes of diagnosis, are the location of the glands, the places of predilection, and the organs which are tributary to them, or to which they are related. Frequently the infection passes through the lymphatic tract unperceived and becomes a concealed infection, producing adenitis, which manifests itself by symptoms of pressure, such as dysphagia, where the retropharyngeal groups and the subeustachian gland is involved; by trismus, when the buccal glands of the cheek are infected; and by torticollis, when the deep cervical group under the sternocleidomastoid muscle are inflamed. The recurrent nerve, if pressed upon by an inflamed tracheal gland, may induce aphonia, as in cases of carcinoma of the esophagus or larynx.

The tonsils, while frequently the seat of primary infection, may become infected secondarily in acute infectious diseases of the neighboring organs, through the lymphatic channels. The same may be true in syphilis and in operative procedure in the nose, especially when tampons are used.

The lymphatic tracts play a more important rôle as carriers of infection than is assigned to them. Through the intimate anastomoses of the lymph channels the infectious material is spread more often than by contiguity or by way of the sanguinary system.

The lymphatic system supplies the material for repair, removes waste, and by mechanical filtration and by phagocytic and lytic action of its lymph constitutes a defense function.

The regression of the lymphatic system with age permits the conclusion that a maximum of danger is offered to the child because of its ready permeability by infectious matter, its richness of network and frequent anastomoses, as compared to the adult; hence the earliest treatment of the affected region of the mouth, nose and throat are indicated.